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THE DIAGNOSIS OF DILATATION OF THE BRONCHI IN CHILDREN BY MEANS OF THE INJECTION OF IODINIZED OIL*

P. ARMAND-DELILLE, M.D.
PARIS, FRANCE
AND
C. F. GELSTON, M.D.
SAN FRANCISCO

The diagnosis of bronchial dilatation in children very often presents the greatest difficulties, not only because the physical signs are particularly difficult to interpret at this age, but because a whole series of affections involving loss of substance or giving rise to abundant expectoration is encountered with greater frequency in young patients.

Bacteriologic examination of the sputum, while permitting the positive diagnosis of tuberculosis, furnishes no other characteristic feature. Until the present time, fluoroscopic and radiographic examinations of the thorax have not given a picture sufficiently clear cut to allow of a topographical diagnosis.

The use of iodinized oil, introduced to radiology by Sicard and Forestier, now permits one an absolutely exact picture of the extent and distribution of the lesions. The first results obtained in the study of the bronchial tree in children are herewith submitted.

Sergent and Cottenot were the first investigators to apply the method of the injection of iodinized oil for the diagnosis of bronchopulmonary conditions in adults. They demonstrated, especially in bronchial affections, that after the intratracheal injection of the oil (a vegetable oil containing 0.54 gm. of iodin to the cubic centimeter), it became particularly easy to determine the exact limits of the healthy or altered bronchial cavities, as well as those of abnormal cavities in the parenchyma of the lung itself.

Their first results strongly attracted our attention, because the problem of certain bronchial dilatations presented themselves with considerable frequency in the service for tuberculous children at the Hospital Debrousse. Fairly often children are sent to this service with the diag-

* Received for publication, July 9, 1924.
* Read before the Thirty-Sixth Annual Meeting of the American Pediatric Society, Pittsfield, Mass., June 5-7, 1924.

nosis of pulmonary tuberculosis who present either the signs of consolidation or of cavity, children frequently treated for a considerable time in spite of the absence of tubercle bacilli in the sputum at repeated examinations, including those of condensation, homogenization and guinea-pig inoculation.

This diagnostic error may occur with greater frequency, as has been insisted on by Rist and Ribadeau-Dumas, due to the fact that it is not at all necessary that a tuberculous cavity should occur only at the apex, particularly in children. They are frequently enough found even at the base. Further, it is not to tuberculosis alone or to dilatation of the bronchi that cavities occupying the middle or lower thirds of the lung are dependent. An interlobar empyema, opening into a bronchus, a pulmonary abscess, an open hydatid cyst, can all provoke the signs of cavity with the appearance of more or less abundant purulent expectoration, but without the presence of tubercle bacilli.

In such cases it is frequently most difficult to secure a sharp outline of the lesions, and therefore, for one to be able to give an exact interpretation. We will lay emphasis a little further in the paper on the lack of information given in many cases by simple fluoroscopy and radioscopy, whereas, injection of iodinized oil furnishes an exact outline of the cavities, thus permitting the establishment of the topography and facilitating a precise diagnosis of the pulmonary cavity, together with the determination of its etiology.

As soon as these points were established by Sergent and Cottenot, we applied their method in children. We wish to thank Dr. Cottenot for having initiated us in his technic, particularly in its adaptation to the type and proportions of the infantile larynx and trachea.

As has been indicated in the foregoing, this mode of investigation renders great service in all cases where one wishes to prove or disprove the presence of a bronchiectasis. As a general rule it is not useful for the topography of tuberculous cavities which radiographically and fluoroscopically give sufficiently sharp images. Nevertheless, we have had occasion in one case to inject an apical cavity. This type of intervention has not been followed by any bad results. Nevertheless, we agree with Sergent that it is not prudent systematically to practice injections in tuberculous patients, because of the chance of causing an increase in congestion due to the influence of the iodin. In those cases, however, where there is a marked contrast between the physical findings and a more or less indefinite radiographic picture which is difficult of interpretation, there may be an indication for the injection of an opaque liquid.

Further, the application of the method of iodinized oil injections is useful in those more or less cachectic patients who are frequently sub-

febrile, and who present wasting and expectoration often of a nummular type, which symptoms are so strongly suggestive of tuberculosis. One is surprised at the inability to demonstrate the bacilli and is therefore obliged to consider the hypothesis of bronchiectasis. On its verification depend future prophylactic measures as well as the prognosis.

In other cases the primary diagnosis may be that of an interlobar pleurisy or a pulmonary abscess. Nevertheless, a low grade but a fairly consistently maintained fever and the slow progress of the disease are suggestive of dilatation of the bronchi. The presence of trophic changes in the distal phlanges terminating in the deformity known as hypertrophic pulmonary osteo-arthropathy may only complicate the diagnosis; other conditions may be causative of this trophic change while on the other hand its evolution may be very rapid in a case of bronchiectasis of comparatively short duration. The past history is frequently enough of no value whatsoever. In short, one hesitates to counsel exploration, for instance in the shape of thoracotomy, and yet there is the fear of deferring investigation for too long a time. It is just these instances where the injection of iodinized oil permits us to gain information and determines the advisability for or against surgical intervention.

To cite further conditions where the injection of iodinized oil is of great value may be mentioned the possible presence of an hepatic, subdiaphragmatic, or perirenal abscess. It is not an easy matter to prove accurately the supradiaphragmatic or infradiaphragmatic position of a cavity or of a collection of pus, when one is confronted by contradictory and multiple physical and radiographic findings. In fact, conditions such as these may, and frequently do, cause such a marked diminution in the transparency of the base of the lung, that there is great difficulty in differentiating hepatic and pulmonary shadows, and in outlining the dome of the diaphragm and the costophrenic sinus.

TECHNIC

For reasons which will be given later, we believe the method of injection through the cricothyroid membrane to be the most satisfactory. Because of a very considerable pressure necessary in injecting the heavy oil, an unusually strong syringe must be employed, similar, for instance, to the Guyon or Record models, and with a capacity of 20 c.c. It should be supplied with a stout rubber tubing, for attachment to the trocar, which will not become distended under pressure and still be thoroughly flexible. The trocar is one especially designed by one of us (A.-D.) for operating on children (Fig. 1). The curved needle of Sergent and Cottenot was at first employed, but its use was discontinued, owing to the fact that children are naturally nervous and excitable under any instrumentation, and therefore, do not restrain coughing or movements of swallowing. The needle may even be displaced as a result and

penetrating the posterior wall of the trachea allow the injection of the oil into the areolar tissue. Several cubic centimeters of oil have thus, on one or two occasions, been injected into the peritracheal cellular tissue. Therefore, in order to prevent this occurrence we have had the before mentioned trocar constructed. It is a curved instrument with a diameter of 1.5 cm., and is made of steel. It is, in fact, made on the principle of the tracheotomy cannula, but it is small enough to obviate the necessity for any considerable anesthesia, and further, prevents damage to vessels and destruction of tissue. The degree of curve and the length of the cannula may be modified according to the age and size of the child, similar to the instruments employed in tracheotomy.

Fig. 1.—Needle for intratracheal injection.

The end of the trocar for attachment to the syringe is fitted with a flat quadrilateral plate approximately 2 cm. in length with a small eyelet at either end. To these are attached cotton tapes which, when tied around the neck, maintain the instrument firmly in place. The stilet of the trocar is constructed with a thick knob to serve as a handle which may be easily grasped and manipulated without disturbing the trocar.

The following points are of importance in preparing for the injections. In the first place the oil must be warmed, either in its original container, or in the syringe in order to render it more fluid. It is wise also to have a syringe prepared containing 2 c.c. and another containing 5 c.c. of a 1 : 100 solution of procain. We also administer 0.005 gm. of morphin sulphate to the child a half hour before instrumentation.

METHOD OF PROCEDURE

The patient is placed in a horizontal position on the fluoroscopy table where he may be immediately roentgenographed without disturbance after the injection of oil. As for a tracheotomy, the head is placed in hyperextension in order to more easily locate the cartilage. A sand bag of appropriate size under the shoulders is the best method of obtaining this hyperextension. At least two assistants are needed in the manipulation.

After having prepared the skin with tincture of iodin, the operator places himself on the right of the patient, immobilizing the thyroid cartilage between the thumb and second finger of the left hand, while with the index finger he palpates the cricothyroid membrane. He then rapidly anesthetizes the skin and subcutaneous layers of tissue with procain, and, guided by the left index finger which has located the correct position, the trocar is forced through the tissues in a plane perpendicular to the larynx. Following this, by a movement upward of the plate at the base, the trocar is pushed farther into the trachea and at the same time becomes parallel with its walls, while the plate rests flat against the neck. When the stilet is withdrawn, a whistling sound of the inspired air denotes a correct position. The syringe containing 5 c.c. of procain is now attached, the head of the patient is slightly withdrawn from its extreme hyperextension in order to prevent the liquid from flowing toward the glottis, and slight pressure exerted to cause its entrance. A forcing out of the piston and the presence of bubbles of air caused by the cough incident to the irritation of the first few drops of the solution confirm the correct position of the trocar in the trachea. Should these indications not be present, one of course suspects a false entry, or the presence of a fold of mucous membrane over the opening. A small amount of manipulation of the cannula and the use of the stilet will usually give results fairly quickly.

The initial coughing rapidly abates with the anesthesia of the tracheal mucous membrane. The operator then attaches the syringe containing the iodinized oil and begins the injection, which should not be too rapid. During the course of the injection or immediately afterward, it is wise to elevate first one side of the patient and then the other in order to facilitate the distribution of the oil. If it is suspected that one side only is affected, the lower position for that side may be maintained entirely. From 8 to 10 c.c. of oil suffices as a rule to inject the bronchial tree of one lung, in children aged from 7 to 14 years, and requires from six to eight minutes or less.

The child is then immediately radiographed in a horizontal position. Fluoroscopy will demonstrate if there has been sufficient oil injected, in

which cases the trocar is withdrawn. There is no hemorrhage and no dressing is needed. Radiographs are then made in the vertical position.

Such is our usual method. It has been suggested that this procedure which we have adopted might be replaced by the supraglottic or transglottic, but whereas an anesthesia of the pharynx and larynx may be fairly easily and satisfactorily secured in the adult, it is a very difficult result to obtain in children, so that as a result of the poor anesthesia, a major portion of the iodinized oil is coughed up and then swallowed. A large part of the oil in fact is thus disposed of in the adult. We have deemed it wise, therefore, to entirely discard any method but that of injection through the cricothyroid membrane.

This method has been practiced without any accident whatsoever, although certain incidents are to be noted in spite of the fact that they are not of a dangerous character.

The first drips of liquid in the trachea provoke an access of coughing which quickly subsides. This reflex is more particularly noticeable in nervous children and causes a reflux of oil into the pharynx, then the esophagus and stomach, causing shadows in the radiographic films which, however, are readily enough located.

Boidin has reported a case of edema of the larynx, attributable to an intoxication from iodin, and counsels a primary investigation into a possible idiosyncrasy to this drug. In our observations this precaution has not been taken and fortunately we have not been obliged to regret it. On a few occasions a certain number of drops of the oil were injected into the perilaryngeal tissue and were perfectly well tolerated. In one case investigated recently at the Children's Hospital, San Francisco, there was a small amount of subcutaneous emphysema in the tissues of the neck for one day.

In one case certain symptoms were observed of a somewhat dramatic character which should be reported. In a very unruly child, aged 14 years, a first attempt at injection was unsuccessful. At the second attempt, made some time afterward, the first contact of the oil caused the phenomena of suffocation, intense dyspnea, barking cough and congestion of the face. We would have feared asphyxia and collapse except for the fact that the pulse remained perfectly firm and regular. It would seem in this case that one might suppose a state of autosuggestion. Nevertheless, shortly afterward, two hemorrhages occurred unquestionably from the pulmonary lesion and not from trauma. The differential diagnosis was of great interest, but it was not thought advisable to make another attempt.

It is well known that iodinized oil injected intramuscularly or subcutaneously is retained for a very appreciable length of time. But in the

respiratory apparatus, once the anesthesia has disappeared, the oil acts as a foreign body and provokes sufficient coughing to cause its prompt expulsion. A small amount remains, however, for some time and must be kept in mind because of the risk to other radiologists not informed of the proceedings, of drawing false conclusions.

The anesthesia does not persist for much longer than ten minutes, and radiography and fluoroscopy should be practised at once after injection. Beyond this length of time the images are much less distinct and can be seen to diminish little by little.

As a control, an injection was made in a child approaching as closely as possible to normal. She presented no abnormal physical findings, although there was a history of measles and whooping cough which might presuppose some small alteration in the bronchial tree. Radiography demonstrated, in fact, small irregularities in the medium and small bronchi of the left lung. In the right lung fields, however, there was a characteristic picture of the normal bronchial tree, namely, the opaque paths extending from the hilum, regular in outline, of small caliber, poorly outlined the farther from the hilum which correspond to the small and medium sized bronchi. There was no evidence of ampulliform, fusiform or circular shadows. The last named are the typical signs of bronchial dilatation. It is wise to recall, nevertheless, that certain of these shadows may give rise to the idea of dilatation, whereas they are in reality a projection of the bronchus directed perpendicularly to the screen, parallel to the rays. They are naturally simply cross sections of a bronchus and not evidence of dilatation. They are to be recognized by their smooth, circular outline, and by the fact that they are surrounded by a zone of normal lung tissue.

In contrast to the foregoing, may be cited the findings in the following case.

This was a patient, aged 13 years, having nothing of significance in the family history and with no history of exposure to tuberculosis. She had had pertussis at the age of 4 years, measles and scarlet fever a little later. At the age of 7 years she suffered from a high degree of persistent fever, which was attributed to pneumonia followed by an empyema. Following this, the patient continued to cough and was sent to the hospital with the diagnosis of pulmonary tuberculosis.

Physical examination at this time showed a retraction of the left half of the thorax, and an extensive area of dulness over the whole of the left lung somewhat less marked on the right. There were, further, coarse moist râles scattered throughout both lungs. There was very marked pectoriloquy at the left base; repeated examination of the sputum was negative for tubercle bacilli.

The child was sent to Brevannes, a sanatorium for tuberculous children, where she remained for one year. At the end of 1922, she returned to her family and then was readmitted to the service at Debrousse, with a card certifying to the fact that "this child is infected with a chronic pulmonary tuberculosis, is contagious, and should be prevented from attending school."

Fig. 2.—Roentgenogram before injection.

The same findings as in 1921 were again demonstrated, namely, dulness at both bases, much more marked on the left, coarse rhonchi throughout both lungs, subcrepitant râles and pectoriloquy at the left base. The temperature was normal, coughing, especially pronounced in the morning, was productive of an abundant white sputum containing no tubercle bacilli. The child had acquired a certain amount of cyanosis. There was considerable clubbing of the fingers and the whole clinical

picture would have substantiated the diagnosis of bronchial dilatation, except for the fact that the roentgenogram showed a marked degree of mottling in the middle portion of the left lung which was much more indicative of tuberculosis.

This lengthy description is submitted simply to draw attention to the difficulty in diagnosis, and from the standpoint of prophylaxis, a positive or negative diagnosis of tuberculosis is extremely important.

With injection of iodinized oil we obtained a particularly striking picture of dilatation of the bronchi. There was shown a series of shadows, each one the size of a pigeon's egg, oval, regular in outline, somewhat superimposed in the horizontal position. In the vertical position, on the contrary, the opaque liquid collects at the bottom of the small cavities, sharply isolated, in this case giving an appearance described by Dr. Darbois of "fleurs de glycenes" (wistaria). The liquid also varies its position in moving the patient which is still greater proof of bronchiectasis.[1]

In viewing these roentgenograms fluoroscopically a most striking idea of the topography of the infected bronchi is obtained. A further observation recently presented to the Society of Pediatrics by Dr. Robert Levy is also of great interest because it is apparently an early stage of the condition which we have just described. The physical signs were less marked, but one could hear almost a gurgling sound at the right base behind (a simple roentgenogram was not at all conclusive, Fig. 2). Injection of iodinized oil shows in the prone position (Fig. 3) many pedunculated cavities the size of a grape near the upper right bronchus and four small round cavities on the left also with pedicles extending to the bronchus, in the vertical position (Fig. 4) as in the preceding observation. There appears a series of crescents with concavities above, while the extremities are continuous with the shadows of the bronchial walls.

Another observation was on a child, aged 8 years, presenting signs of consolidation at the right base, with evidence of probable bronchial dilatation in the left middle and lower areas of the lung. The sputum on repeated examination was negative for tubercle bacilli. There was moderate emaciation and a low grade temperature. Injection confirmed very distinctly the evidence of bronchial dilatation on the left with approximately normal findings in the upper portion of the right lung (Fig. 5). There was, however, some impediment, apparently, to the entry of the oil into the lower lobe on the right, as is very clearly seen. A further injection of 5 c.c. of oil was made, with the idea of

1. Other photographs illustrating the cited cases are to be found in an article by Armand-Delille, P.: Presse méd., Paris, **32**:421 (May 14) 1924.

Fig. 3.—After injection, prone position.

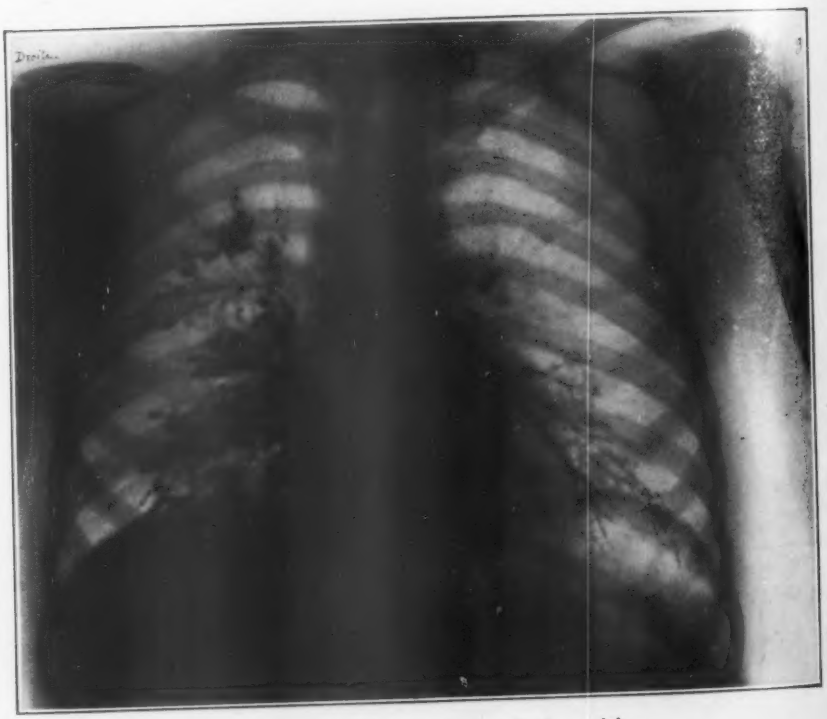

Fig. 4.—After injection, vertical position.

either confirming this obstruction as permanent or as simply a temporary block. Figure 6 shows the presence of the oil in the major bronchus leading to this area to a greater distance, and apparently in some small bronchi behind it, but the area is definitely limited in the middle and upper portion of the lower right lobe and is unquestionably due to a

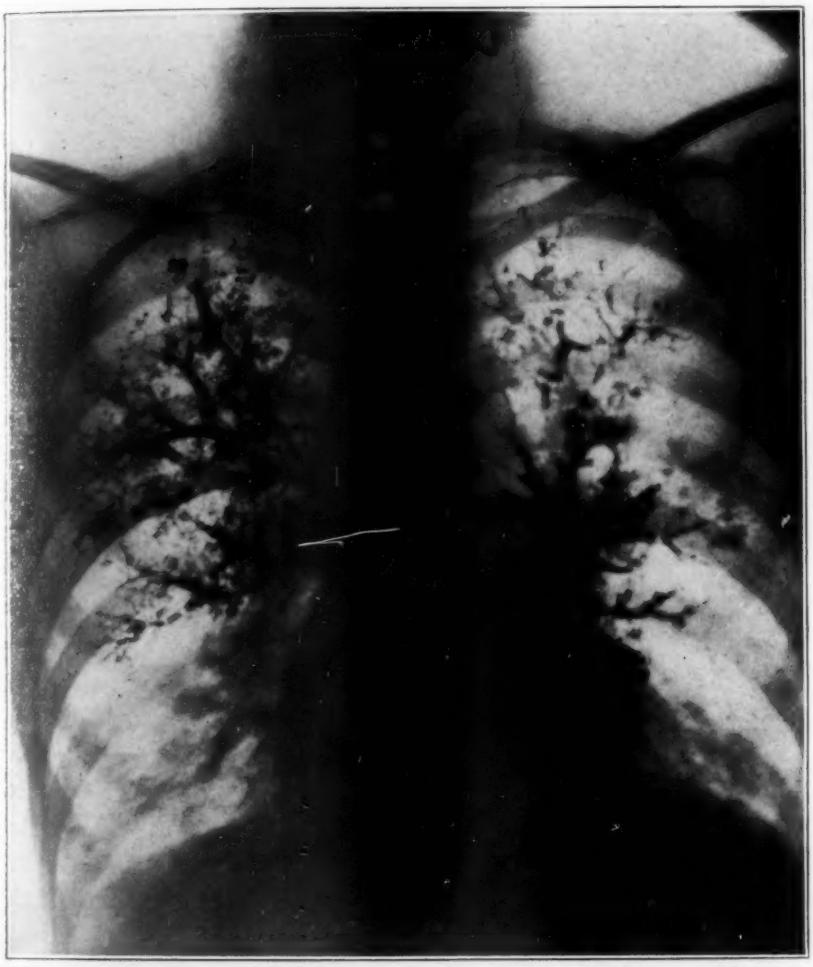

Fig. 5.—Injection of oil; prone position. Dilatations of left base. Poor entry of oil in lower right bronchial tree.

fibrotic, sclerotic healing of a lesion with permanent narrowing and occlusion of the bronchi.

It is not necessary that the lesions should be very marked in order that the investigation by means of iodinized oil should be of interest and

value. Even cases of small multiple dilatations, following pertussis, can give characteristic images. An example was that of a girl, aged 7 years, sent to the hospital with a diagnosis of pulmonary tuberculosis. At first sight this diagnosis seemed rational, as the child had been in prolonged contact with her sister, who had died from pulmonary tuberculosis.

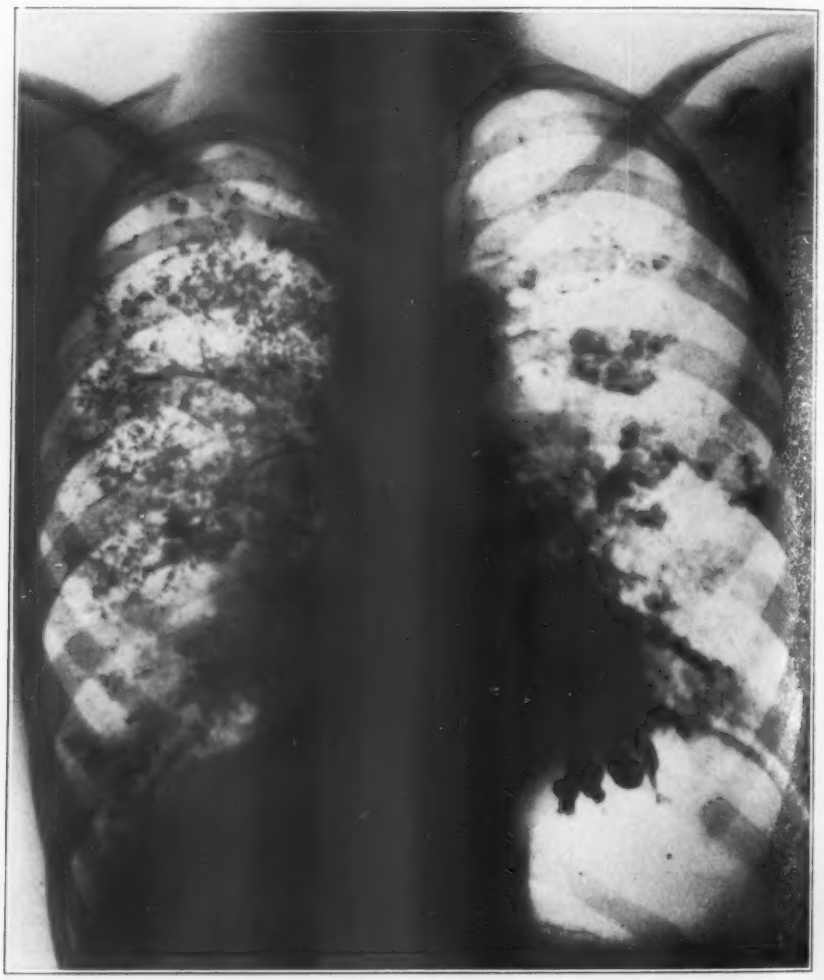

Fig. 6.—Same case, prone position. Further injection of oil forced a certain amount into the bronchi of the right lower lobe.

Contradiction to the diagnosis was, however, found in the physical findings, predominantly at the right base; the general condition of the child, the negative Pirquet reaction and the repeatedly negative sputum examinations.

A first roentgenogram showed normal pulmonary parenchyma, another taken after the injection of iodinized oil showed, on the contrary, a series of small dilatations confined to the bronchial ramifications to the exclusion of the larger bronchi.

The injection of iodinized oil was of much value in another child, aged 8 years; in the neighborhood of the middle and upper lobe of the right lung in back were signs of cavity formation. There were gurgling râles and diminution in the sounds of coughing. Tuberculosis could not be demonstrated. Radiography after an iodinized oil injection demonstrated ampulliform dilatations located near the hilum on the right.

In the cases cited, the use of the iodinized oil injections was of great value in proving the presence of bronchial dilatation, determining its extent and location. With further experience, perhaps, we will be able to render a more exact prognosis depending on the appearance of the various bronchi, and also perhaps to draw from the knowledge gained some deductions regarding therapeutics. Further, even in cases of developing tuberculosis where the diagnosis of bronchial dilatation might be made, this method of investigation can render great service in elucidating the nature of the parenchymatous lesions where, because of the symptoms, there may be a great similarity to a condition of bronchiectasis.

This is demonstrated in the case of a child, aged 11 years, who presented the symptoms and signs of cyanosis, trophic changes in the phalanges, together with profuse pseudo vomiting, i. e., of swallowed sputum, the whole picture more or less justifying a diagnosis of pulmonary tuberculosis because of which the child was sent to the hospital. Clinical examination revealed at the right base evidence of consolidation, with also the signs of a certain amount of fluid. The temperature remained normal. Tubercle bacilli were not discovered in the sputum and on fluoroscopy the lung appeared opaque in the lower half, while under this zone of opacity there was an area less obscure which seemed to indicate a loss of tissue. Examination by means of iodinized oil served to eliminate the diagnosis of bronchial dilatation, and instead demonstrated a supradiaphragmatic cavity which, together with Dr. Sergent, we have interpreted as the result of a pulmonary abscess. The iodinized oil formed a very large, distinctly localized spot on the plate which was taken in October 1923. At a recent examination in February 1924, there was still evidence of the oil (no photograph).

It is of note and interest that in this particular patient there was distinct amelioration of the vomiting, less expectoration, a marked lessening and localizing of the physical signs together with a general

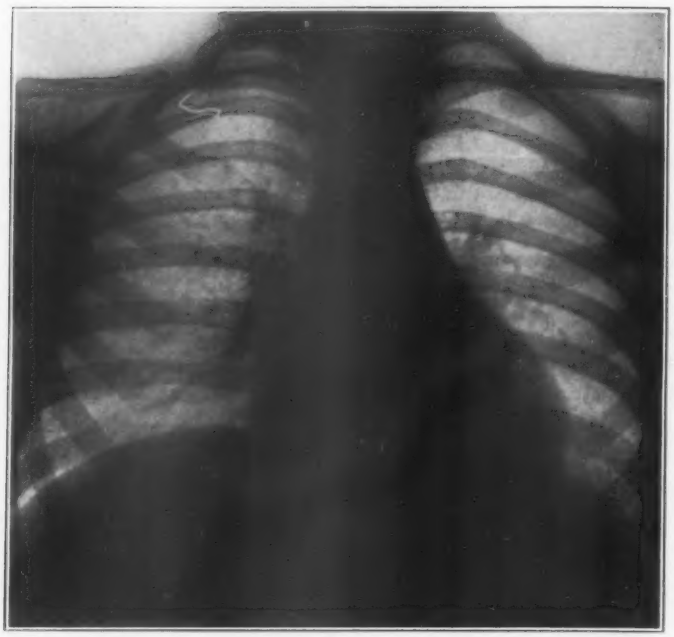

Fig. 7.—Roentgenogram before injection.

Fig. 8.—Small amount of dilatation, especially behind the heart.

improvement in the child's physical condition. We are permitted to hope that this may perhaps have been due to the prolonged presence of the iodinized oil.

In the case of the patient before mentioned, there was no question about the diagnosis of supradiaphragmatic abscess. But this is not always so by any means. The difficulty in localizing a suppurative

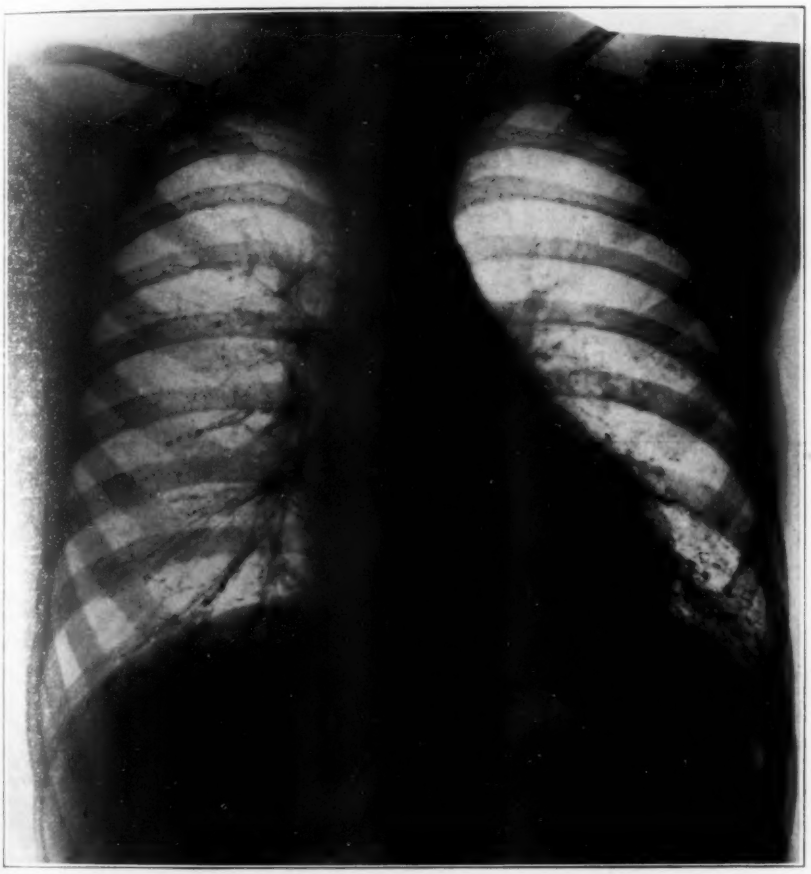

Fig. 9.—The grouping of cavities.

process near the diaphragm is well known. There again may be established the value of iodinized oil in localizing a fistulous tract or a pulmonary abscess.

We feel very regretful, indeed, because of the extremely valuable information which we have derived from our investigation with iodinized oil that we were not able to apply it in numerous cases previously encountered which presented many diagnostic difficulties; for instance,

Fig. 10.—Roentgenogram before injection.

Fig. 11.—Great number of small dilatations, bilateral.

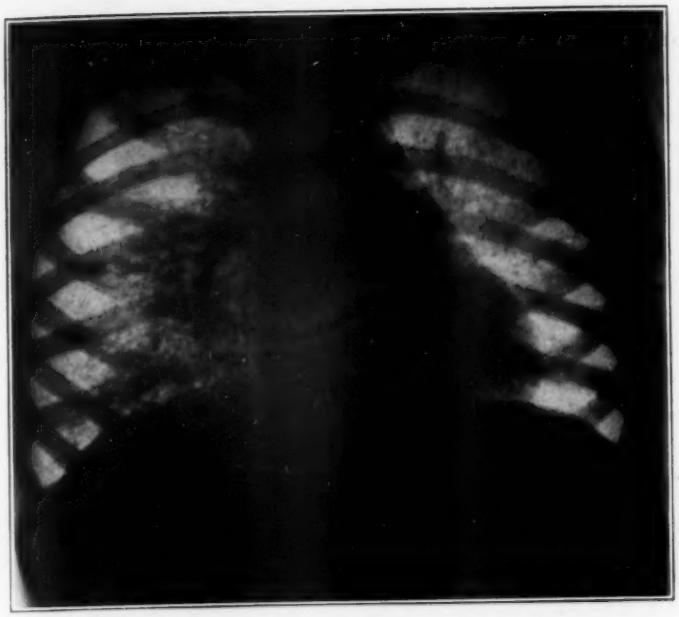

Fig. 12.—Taken ten minutes later, showing greater involvement on right.

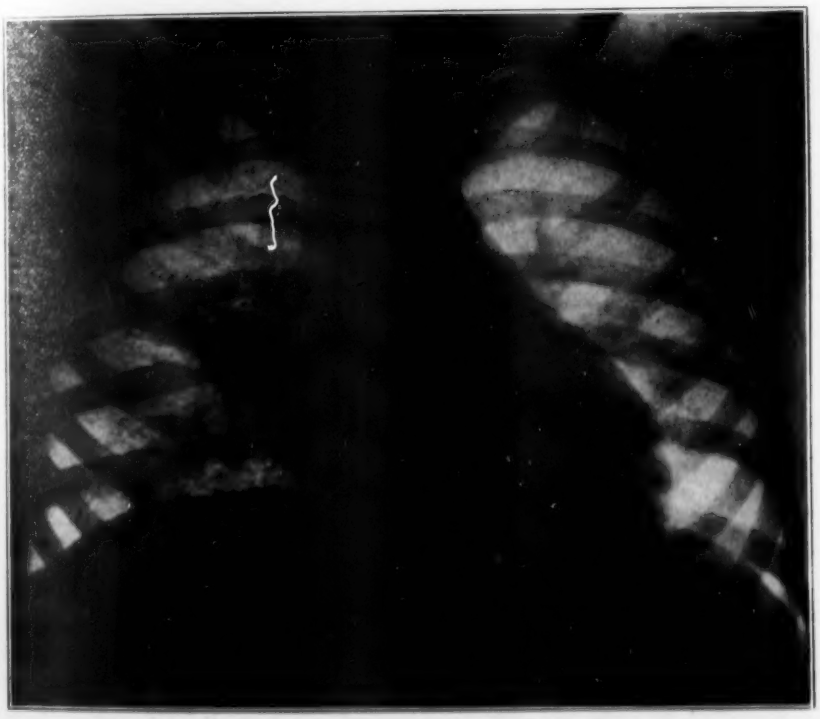

Fig. 13.—Plate taken ten days after injection, showing presence of oil especially on the right.

certain patients showing signs of cavity, without a positive sputum and in which the clinical progress did not at all correspond to a pulmonary infection with tuberculosis. In such cases, the simple roentgenogram showing vague shadows, perhaps indicating loss of parenchymatous tissue is frequently misleading, whereas the iodinized oil gives such clearly cut pictures that all doubt is eliminated.

The observations in a boy, aged 4 years, presenting a history of asthma and repeated attacks of bronchitis are interesting from the standpoint of eliminating any great degree of dilatation. Figure 7 shows

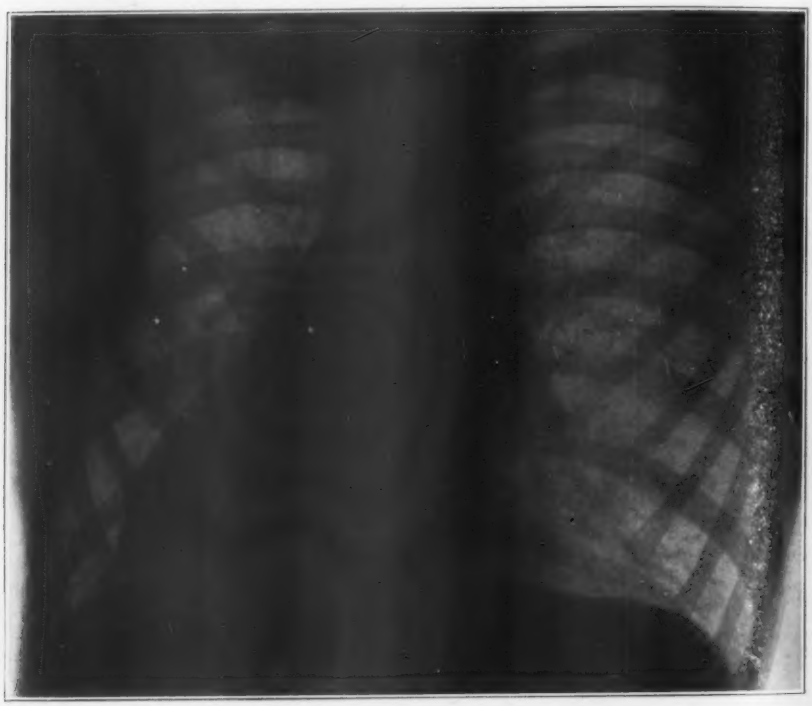

Fig. 14.—Roentgenogram before injection.

the roentgenogram before injection. Figures 8 and 9 indicate the bronchial tree with considerable distinctness, and illustrate particularly some cylindric dilatations in the post cardiac area.

Figure 10 illustrates the findings before injection in a child, aged 3 years, with the history of chronic bronchitis, much sputum and "pseudo vomiting" of the French clinicians. Physical examination revealed many coarse, medium and fine mucous râles throughout both lungs, especially in the interscapular area. Injection of oil demonstrated the presence of a very large number of small dilatations in the

hilum region of both lungs (Figs. 11 and 12). It is of considerable interest from the therapeutic aspect of this study that a roentgenogram, taken ten days after the injection, showed a large amount of the oil in the small cavities (Fig. 13). This is not the case when no dilatation

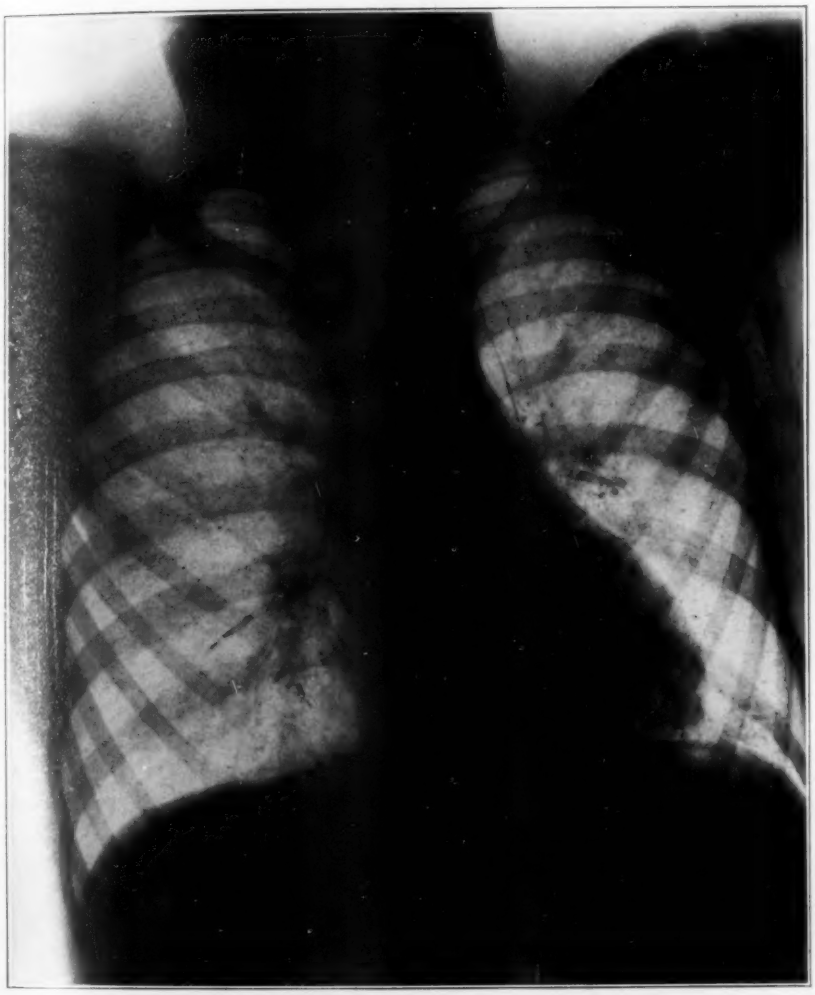

Fig. 15.—Large fingerlike dilatations of bronchi behind the heart.

of the bronchi is discovered. In most such cases all of the oil is expectorated in the course of a few days.

A particularly interesting patient was a boy, aged 8 years, who has been under observation for two years with the physical findings of bronchiectatic cavity at the left base. This has apparently lessened in

size since he was first seen, and the boy's physical condition generally is quite satisfactory, in spite of the fact that there is still considerable coughing and expectoration. Figure 14 shows the radiographic findings before injection. Figures 15 and 16 illustrate clearly the larger finger-like dilatations of the bronchi in the post cardiac area. This child will furnish an excellent subject for observation of the therapeutic result of the iodin.

Fig. 16.—The grouping of cavities, thus explaining the physical findings.

As a final case in point, attention should be given to Figure 17. This child, more or less cachectic, had suffered an extensive involvement of the right lung one year before entering the hospital. Our physical findings demonstrated complete dulness throughout the right chest with, however, the presence of innumerable râles of all types somewhat more marked over the middle lobe. The original condition we ascribed to what we have designated as a

splenopneumonia. There was a very low grade temperature, emaciation, some clubbing of the fingers and toes, and a profuse expectoration. A simple roentgenogram furnished us with little information; one can readily see the findings when iodinized oil was injected. There was a very large multilobulated cavity, probably from bronchial dilatation.

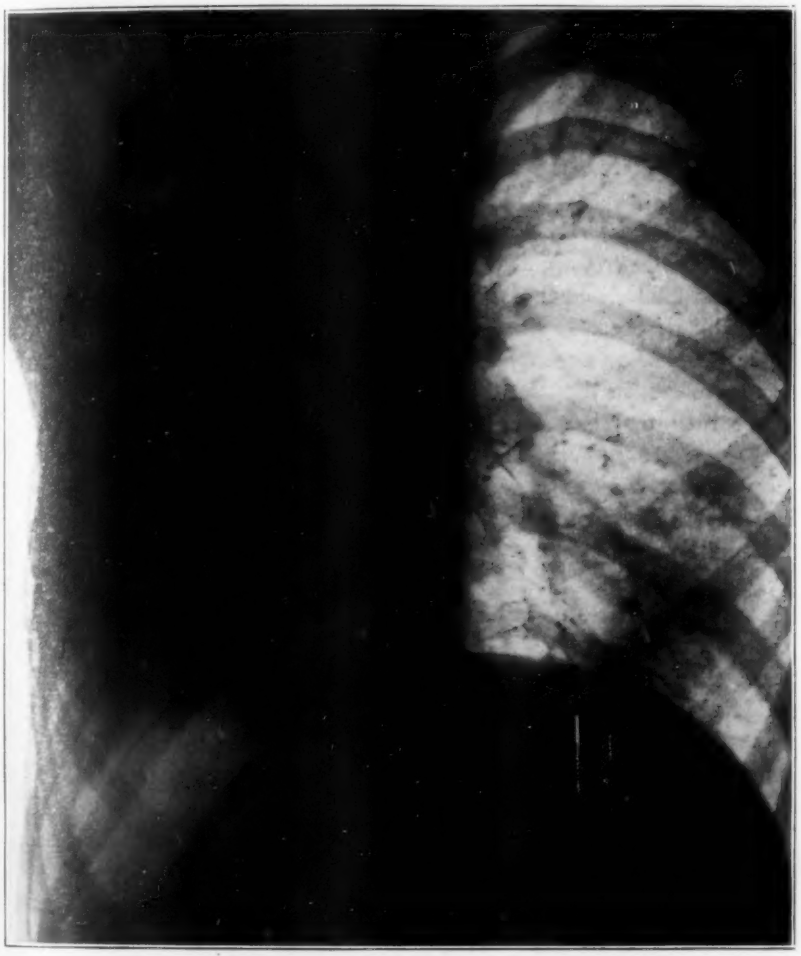

Fig. 17.—The value of injection in localizing lesions, the ordinary roentgenogram being of little aid, due to the thickened pleura in cases similar to the one cited.

CONCLUSIONS

In conclusion, we wish to add a few remarks relative to the method employed and to the benefit perhaps to be derived from it. For instance, utterly aside from any other consideration, the shadow given

in a roentgenogram is of great interest. The contrast, in fact, between other radiographic reproductions and those obtained by means of the injection of iodinized oil offers extremely valuable information and should permit of the following conclusions.

If there is the picture of multiple shadows in the neighborhood of the hilum with an excessive bronchial ramification, the shadows being joined to the bronchi by a pedicle, the diagnosis of bronchial dilatation is justified. It may be demonstrated later, perhaps, that this conclusion must be modified in cases of glandular cavities, but until the present no such cases have been encountered.

If the shadows are confluent but can, nevertheless, be separated by careful examination, the diagnosis of dilatation of the bronchi is again established.

If the larger bronchial ramifications, without showing bunched localized shadows, appear noticeably enlarged one must admit a cylindric dilatation of the bronchi.

Finally, if there is simply a question of shadows along the smaller bronchial ramifications, when they are compared to the normal picture, one may establish the presence of a moderate degree of bronchiectasis.

We may disregard tuberculous cavities which are easily enough diagnosticated by means of ordinary roentgenograms. It may be noted, however, that cavities left by pulmonary abscesses are usually located far from the hilum and the mediastinum, above the diaphragm, and are usually single.

Finally, aside from the great diagnostic interest presented by this method of intratracheal injections of iodinized oil in children, emphasis should be placed on the fact that it is entirely innocuous and further holds out the hope of perhaps having a therapeutic application of some considerable value. In fact, we have observed in a number of young patients a marked lessening in cough and expectoration, together with modification of physical signs indicating a diminution in the size of the bronchial cavities. It would appear that there might be a local effect of the iodin. So far, we have not checked this by means of second injections.

From all standpoints, it seems to us that this method, having already been proved efficient and of no danger, promises to furnish us with valuable and important information regarding the normal and pathologic appearance of the bronchial tree in children.

NEW METHODS FOR MAKING ESOPHAGEAL STRICTURES VISIBLE AND PHOTOGRAPHING THEM

PARTICULARLY IN THE CASE OF CHILDREN *

ARTHUR POLLERMAN, M.D.
NEW YORK

The walls of the esophagus are easily penetrated by the roentgen rays and so do not show any shadows even to the eyes of expert roentgenologists. Indirectly, however, by introducing materials which cast shadows into the esophagus, the outlines may be made visible. This method does not yield a complete outline because the peristalsis carries the material away from portions of the esophagus.

Up to the present, the most acceptable, and almost universal, technic has been to mix bismuth or barium compounds into a paste with water, or some other palatable medium.

In the case of very young children, however, their natural resistance almost always throws obstacles in the way of the application of such a paste. In an effort to avoid the difficulties of this technic I devised the following two methods.

Previous to the war I had rubber tubes of different sizes made for me, with thin walls impregnated with as much lead as the rubber would carry without losing its elasticity. These tubes are sealed at one end and may easily be inflated. One of these tubes is drawn over a well oiled bougie and inserted into the esophagus, after which the bougie is withdrawn and the tube inflated. In consequence of the inflation, the walls of the tube conform closely to the walls of the esophagus and owing to the contained lead permit the observation of the esophageal walls and of any strictures or other deformities which are present.

In the case of young children it is found to be more convenient to inflate the tube without withdrawing the bougie.

This method is easy and convenient and of advantage in most cases, excepting in those in which there is a very narrow stricture or in which there is found a very great diverticulum. By this method the esophagus is well outlined and may be photographed even in a horizontal position. The inflated tube shows the precise shape all along the esophagus, not only the principle stricture, but if there are others, it shows all of them in detail.

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* Received for publication, Aug. 9, 1924.

* Details from the Children's University Clinic connected with the Stephania Children's Hospital in Budapest, Hungary. Director: Dr. John Bokay, Professor of the University.

In the cases in which the tube cannot be used, an alternative technic has been devised by Dr. Veli, also a physician of our clinic, which has proved to be superior to the use of bismuth in water. This consists

Fig. 1.—A boy, aged 6, drank lye in August, 1921. The roentgenogram was taken in May, 1924.

in the administration of a suspension of bismuth subcarbonate in an emulsion [1] composed of: oil of bitter almond, 8 parts; gum arabic 4 parts, and water, 8 parts. By vigorously shaking the bismuth with

1. In case of children, simple syrup (1 part to 10 parts of the emulsion) is usually added before administration.

Figure 2

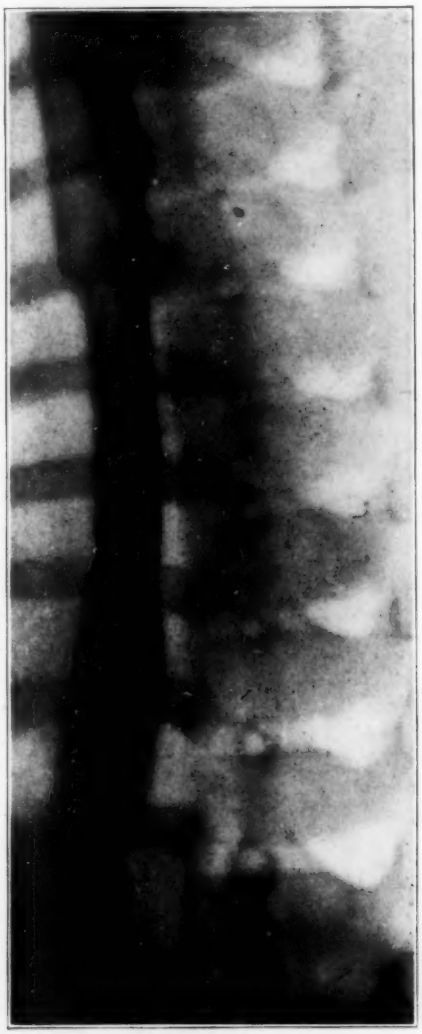

Figure 3

Fig. 2.—A boy, aged 13, drank lye in July, 1923. The roentgenogram was taken in May, 1924.

Fig. 3.—A boy, aged 4, drank lye in October, 1923. The roentgenogram was taken in May, 1924.

this medium in a test tube, a suspension is obtained which yields an easily perceptible and very even shadow, and which is not displaced so quickly by the peristalsis of the esophagus. This mixture shows the walls of the diverticulum of the esophagus as well as the narrow lumen of the stricture.

Lastly, it is not necessary to use as much of this mixture as of the water paste, experience having shown that as little as one or two teaspoonfuls are sufficient in most cases.

HYPOGLYCEMIA AND ACIDOSIS IN FASTING CHILDREN WITH IDIOPATHIC EPILEPSY*

EDWARD B. SHAW, M.D.
SAN FRANCISCO
AND
MARGARET MORIARTY, S.B.
BOSTON

Fasting of late has been frequently employed in the therapeutics of epilepsy, and numerous reports of fasts for this purpose are found in the literature. In the majority of cases, improvement of at least a transient nature follows fasting but permanent cure has not been demonstrated. Thus far, no consistent explanation has been advanced for the improvement which occurs.

During the last two years, nineteen fasts have been carried out on epileptic children in this clinic. During the fast these cases have been studied, in order to determine the effect of fasting on the metabolism, with the hope that some constant change might serve to explain the improvement of the epileptic symptoms during the fast and perhaps form a rational basis for further treatment.

Studies carried out in some of these cases have formed the basis of a previous report,[1] and it is the purpose of this paper to present certain more or less significant findings as exemplified in a series of five additional cases, which serve to extend and corroborate the previously reported data.

The cases which comprise this series varied in the degree of their symptomatology from mild to moderately severe epilepsy. There was little in the history, the physical examination, or the findings in laboratory studies carried out before the fast which showed departure from the normal other than the occurrence of epileptiform seizures. Briefly, the individual status was as follows:

REPORT OF CASES

CASE 1.—J. B., a girl, aged 9 years, whose parentage was excellent physically and mentally; there was no familial taint of any kind. Her early history was uneventful. Epileptic attacks commenced four years before entry to this hos-

* Received for publication, Aug. 12, 1924.

* From Children's Medical Department, Massachusetts General Hospital, under the direction of Prof. Fritz B. Talbot.

* This article is No. 27 of a series of studies in metabolism from the Harvard Medical School and allied hospitals. The expenses of this investigation have been defrayed, in part, by a grant from the Proctor Fund of the Harvard Medical School for the study of chronic diseases.

1. Hoeffel, Gerald, and Moriarty, Margaret: The Effects of Fasting on the Metabolism of Epileptic Children, AM. J. DIS. CHILD. **28**:16 (July) 1924.

pital, and always consisted of attacks of momentary unconsciousness, rolling of the eyes, twitching of eyelids, and hyperextension of the neck. If she was standing at the time of attack she fell, but more extensive attacks never developed. Consciousness was regained almost immediately and the child usually continued what she was doing just previous to the onset of the seizure. The patient had been the subject, six and four months before this entry, of two of the previously reported fasts,[1] which had brought about distinct improvement for a time. When she entered the hospital she was having five or six attacks daily. Physical examination revealed nothing of significance except an internal strabismus and very poor bodily mechanics, which were corrected before the fast was instituted. Physical development was good for her age. The urine, blood and blood Wassermann tests were negative.

CASE 2.—W. P., a boy, aged 11 years, was of Jewish parentage. There was no familial taint. Except for the usual infections of childhood, his previous history was not significant aside from convulsions accompanying otitis media at the age of 1½ years. There was no recurrence until nine months before this entry, when the patient commenced to have characteristic epileptiform attacks at the rate of four or five a week to two or three daily. An aura was usually present consisting of ringing in the ears and numbness of the right arm, and these symptoms were frequently noted without developing into convulsions. A previous fast had resulted in temporary improvement. Physically, the boy was well developed and nourished and was mentally bright. General physical and neurologic examinations were entirely negative. The urine, blood and blood Wassermann tests were negative.

CASE 3.—C. P., a boy, aged 13 years, was of Italian parentage. The family history was not relevant. For the last year the patient had had frequent attacks, characterized by sudden loss of consciousness, falling, but without muscular spasm or loss of sphincter control. The attacks lasted for a few moments and were followed by prolonged stupor. Physically, the child was well developed. Signs of sexual maturity were not developed. The pupils were irregular and reacted sluggishly. The tonsils were markedly enlarged. There were no changes in tendon reflexes. Mentally, he was much below par and failed to cooperate well during the fast. During the first five days of the fast he succeeded in obtaining small amounts of bread and perhaps of other food.

CASE 4.—H. A., a boy, aged 13 years. The father and sister of his mother had been epileptic. He had had the usual diseases of childhood, also repeated attacks of tonsillitis for which tonsillectomy had been done. Since the age of 7 years he had had convulsive seizures of varying severity beginning on the right side, and usually proceeding to generalized convulsions with loss of consciousness and sphincter control. There had been intervals of several months when he was free from attacks. He was of normal height for age but was thin and undernourished and had marked "fatigue type" posture. Signs of sexual maturity were not present. Otherwise his general physical examination was negative. The urine, blood, blood Wassermann tests and roentgen-ray examination of the skull were negative.

CASE 5.—P. G., a girl, aged 11 years, Italian. The family history was negative. The previous history was not significant, aside from the occurrence of frequent attacks of sore throat. Tonsillectomy had been done nine months before entry to our wards. The attacks were said to have begun exactly seven weeks after tonsillectomy, occurring almost regularly every eight days. Momentary aura preceded the attacks, which consisted of generalized convulsions with loss of sphincter control. Usually four or five attacks occurred at intervals of a few hours. Her general development was excellent; sexual maturity had not been attained. There were no significant physical findings and her mentality was unimpaired. The urine, blood and blood Wassermann tests were negative.

So far as possible, the fasts in these children were conducted under uniform conditions. During a preliminary period, the child was kept at rest in bed and fed a weighed diet of constant protein, carbohydrate and fat composition, and control observations were made on the factors which were to be studied during the fast. In all except one case this preliminary period consisted of three or more days. The fast on W. P. (Case 2) was commenced after one day's study, previous observations having given normal control findings.

The duration of the fast was from ten to fourteen days. During this period nothing was given except water, which was administered to a minimum of 75 c.c. per kilogram of body weight every twenty-four hours. In some of the earliest fasts (1921) it had been feared that severe symptoms of acidosis might appear under this regimen, so glucose and orange juice were given as a preventative, but the clinical results were unsatisfactory. Later developments showed that if water were administered in large quantities severe symptoms of acidosis did not develop, and peculiarly more improvement seemed to result if only water were given. It could be definitely shown that when the fluid intake fell below this minimum, the symptoms of acid intoxication were strikingly increased. Both J. B. (Case 1) and W. P. (Case 2) volunteered the information that they felt much more discomfort from the fast when they took less than the prescribed amount of water, and their appearance bore out this statement.

Many studies were made which are not reported here. In most of these patients observations were made on the blood count, volume of corpuscles, serum albumin, blood pressure, blood calcium and phosphorus. Discussion is limited to certain factors which were observed daily, in order to follow carefully any changes which might occur during the starvation period.

BLOOD URIC ACID

There have been numerous suggestions that an increase in nitrogenous constituents in the blood might be responsible for epileptic convulsions, and it has even been stated that an increased uric acid concentration is responsible for the convulsion. It has been found by some observers that the blood uric acid concentration is increased after attacks.[2] Interest was naturally directed to the effect of fasting on the uric acid concentration in the blood.

Lennox,[3] in extensive observations on both normal and epileptic adults who were fasting, has demonstrated a great increase in blood uric acid during the fast and has carried on some investigations in an effort to explain this.

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2. Romberg and O'Connor: Unpublished work in this Clinic, 1921-1922.
3. Lennox, W. G.: Increase of Uric Acid in the Blood During Prolonged Starvation, J. A. M. A. **82**:602 (Feb. 23) 1924.

In this series, daily estimations of the uric acid in the blood were made by the modified method of Folin-Benedict.[4]

As is shown in Table 1, a marked increase in uric acid concentration was found in all these cases; the maximum increase was about 300 per cent. over the prefasting control. Lennox has discussed the possible explanations for this increase so they need not be commented on here. It is, of course, possible that this increase is accounted for by unrecognized errors in the method of determination. It is apparent, however, that if fasting is accompanied by a true increase in the uric acid concentration that this is incompatible with the improvement which follows if there is any causal connection existing between increased uric acid and the onset of attacks.

Occasional observations in these and other fasting cases have shown no departure from the normal in the creatinin, amino-acid and nonprotein nitrogen constituents of the blood.

CHANGES IN ACID BASE RELATIONS

Other investigators have established the fact that during the fast, glycogen reserves are rapidly used up, after which the source of energy is chiefly from fat. Deficient carbohydrate combustion is attended by imperfect fat combustion, and acetone and ketone acids are thereby produced. The ketone acids unite with the available alkali in the blood[5] and thus reduce the ability of the blood to transport carbon dioxid. These acids are excreted in the urine, while the acetone is eliminated through the urine and the breath.

During these fasting periods the acetone production was estimated by a roughly quantitative determination of the excretion in the breath. The child was asked to blow steadily through 10 c.c. of Scott-Wilson reagent[6] and the number of seconds when clouding first appeared was noted (normally, no clouding appears in five minutes). This gives at best only a general indication of the amount of acetone produced, and it is designed to show only the general tendency of acetone production.

Table 2 shows the appearance time of precipitation in the Scott-Wilson reagent. It is shown that early in the fast the breath acetone rapidly increases to a maximum and tends slightly to diminish toward the end of the second week.

While the acetone was only roughly estimated, the alkali reserve was more accurately quantitated by measuring the carbon dioxid com-

4. Folin, Otto: System of Blood Analysis: Revision of Folin-Benedict Method for Determining Uric Acid, J. Biol. Chem. **54**:153 (Oct.) 1922.

5. Gamble, J. L.; Ross, S. G., and Tisdall, F. F.: Study of Acidosis Due to Ketone Acids, Tr. Am. Ped. Soc. **34**:289, 1922.

6. Mathews, A. P.: Physiological Chemistry, Ed. 3, New York, William Wood & Company, 1921, p. 1118.

TABLE 1.—*Blood Uric Acid (Milligrams per Hundred Cubic Centimeters)*

Case	Control before			Days of Fast													Control after			
				1	2	3	4	5	6	7	8	9	10	11	12	13	14				
1. J. B.	2.88	4.08	3.04	....	....	9.76	12.5	8.96	....	9.6	....	....	8.08	....	7.62	Fast ended		....	2.16	....	3.12
2. W. P.	....	....	2.76	....	4.04	5.16	5.92	9.76	7.12	7.92	6.48	6.04	7.20	6.24	5.80	5.16	7.60	2.80	....	2.80	....
3. C. P.	1.80	....	2.40	....	3.04	4.64	4.88	....	5.88	6.56	6.44	5.60	8.08	4.72	4.84	5.76	5.20	2.32	....	2.72	....
4. H. A.	5.20	3.65	3.36	2.92	3.40	5.20	8.16	9.40	8.16	10.08	9.28	7.92	7.02		Fast ended			5.40	....	....	....
5. P. G.	....	2.28	....	2.80	3.16	5.20	7.84	8.96	7.84	7.00	8.16	7.60	4.32		Fast ended			4.68	2 92	....	2.44

TABLE 2.—*Breath Acetone. Appearance Time in Seconds of Cloudiness in Scott-Wilson Reagent*

Case	Days of Fast													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. J. B.	..	35	20	14	15	23	14	17	12	12	12	14	Fast ended	
2. W. P.	..	22	13	12	17	17	14	12	12	18	20	18	..	20
3. C. P.	..	35	15	28	..	27	22	23	12	18	25	18	20	16
4. H. A.	150	30	25	23	23	17	22	23	22	20	20	Fast ended		
5. P. G.	50	22	17	14	14	14	15	12	18	20	..	Fast ended		

bining power of the whole blood or plasma. The amount of change of the alkali reserve in cases in which determinations were made with whole blood is not directly comparable to those cases in which plasma was used. The change from day to day is consistently indicated, however, by either method. The carbon dioxid combining power was determined daily with blood drawn from an arm vein without stasis and placed immediately under oil.

Reference to Table 3 shows that during the early days of the fast, the alkali reserve reached a minimum on the third to sixth day and remained low throughout the greater portion of the fast; but showed a distinct tendency to rise during the second week returning nearly to normal by the end of the fast in some of the cases.

The carbon dioxid content of the alveolar air was also determined throughout the fast. Specimens were collected in a Haldane tube and were analyzed by the Haldane method. The collection of specimens of alveolar air from children is difficult to carry out and in many cases results are hardly more than an approximation. In the case of cooperative children (as for example, J. B., who had had training in two previous fasts) check determinations were readily made, but on the other hand in some cases (as P. G.) the alveolar specimens were so poor that the results were without significance.

Table 4 shows that during the fast a rapid fall in the alveolar carbon dioxid tension occurs somewhat similar to the drop in the alkali reserve.

HYDROGEN-ION CONCENTRATION

Alkali reserve and alveolar carbon dioxid being accurately determined, it is possible to compute the hydrogen-ion concentration from these data by means of the Henderson-Hassalbach formula. In cases previously reported from this clinic, it was shown that hydrogen-ion concentrations computed in this way underwent a demonstrable rise (fall in p_H) accompanying the fall in alkali reserve.

It is feasible to compute p_H in this way only if the child is cooperative and accurate specimens of alveolar air can be secured. On the first of these patients, J. B., good alveolar carbon dioxid specimens were procurable, and determinations of the p_H by the Henderson-Hassalbach formula gave results which checked.

The hydrogen-ion concentration estimated by this method in all probability demonstrates accurately the degree of shift in the p_H, but for absolute readings this method requires additional correction factors or tonometer analyses of the carbon dioxid combining power.

In order to study more accurately, as well as more simply and directly, the p_H changes in the blood which accompanied the fall in

TABLE 3.—*Alkali Reserve* *

Case	Control before			Days of Fast													Control after			
				1	2	3	4	5	6	7	8	9	10	11	12	13	14				
1. J. B.†	....	....	63.3	....	....	34.5	39.3	36.4	....	35.9	....	....	35.6	....	38.7	Fast ended		....	70.0	....	57.7
2. W. P.‡	....	....	48.0	....	40.5	38.8	34.4	43.4	44.1	41.7	35.6	44.4	43.6	51.0	51.8	50.0	50.5	72.0	....	59.3	....
3. C. P.‡	....	....	60.6	....	44.3	34.5	35.3	stole food		46.6	36.8	39.5	48.1	45.3	46.3	48.0	50.0	56.4	63.7	....	56.8	....
4. H. A.†	64.6	69.6	64.9	64.0	66.2	51.6	51.7	45.7	50.9	45.9	48.2	47.0	46.3		Fast ended				66.3	....	....	....
5. P. G.‡	....	....	49.7	51.9	48.0	....	44.5	47.1	43.5	47.0	45.6	47.8	49.9		Fast ended				57.9	62.2	....	61.7

* All blood samples in the prefasting period were drawn from twelve to fifteen hours after food. Determinations of blood carbon dioxid were made by the method of Van Slyke equilibrating the blood at room temperature with our own alveolar carbon dioxid.

† Carbon dioxid combining power of blood plasma.

‡ Carbon dioxid combining power of whole blood.

TABLE 4.—*Alveolar Air Carbon Dioxid Tension in Millimeters of Mercury*

Case	Control before			Days of Fast														Control after
				1	2	3	4	5	6	7	8	9	10	11	12	13	14	
1. J. B.	....	....	42.9	....	36.3	28.8	31.7	29.0	30.5	30.4	....	29.8	....	....	26.5	28.5	Fast ended	43.4
2. W. P.	....	....	35.3	....	31.5	26.3	24.3	27.5	25.4	26.1	27.8	28.8	27.1	27.4	....	....	27.8	38.4
3. C. P.	....	....	38.2	....	32.0	30.3	39.1	....	36.9	25.7	23.5	25.6	....	....	....	....	23.7	31.9
4. H. A.	40.8	35.9	38.0	38.3	38.1	33.7	31.7	26.1	27.3	23.2	24.4	22.3	24.1		Fast ended			27.5

alkali reserve, the colorimetric method [7] of Cullen [8] was employed in Cases 4 and 5. Standard buffer solutions were checked by means of the potentiometer.

Table 5 shows the changes in p_H which occurred during fasting in these cases. It was originally our purpose to determine the p_H both colorimetrically and according to the Henderson-Hassalbach formula in the last two cases but in these, as in the two preceding cases, we did not feel that the alveolar specimens were sufficiently reliable to give accurate p_H determinations.

It is shown that a definite fall in p_H accompanies starvation in these children. In the case of J. B. (Case 1) the maximum drop in p_H was 0.10, from a p_H of 7.40 to 7.30. Colorimetric determinations showed a fall from p_H 7.42 to p_H 7.32 in H. A. (Case 4), and from 7.45 to 7.32 in P. G. (Case 5).

After reaching the minimum early in the fast, from the third to the fifth day, there was a steady return to approximately normal values for the patient before the end of the fast (from ten to fourteen days).

Recently, Barr, Himwich and Green [9] have demonstrated that changes in the p_H occur in a person under physiologic conditions, such as severe muscular exertion, food, and so on. The change in p_H which occurred in the reported cases was not greater than could have been produced by these physiologic processes, but it should be noted that all of our determinations were made at the same time in the morning on a fasting child who was at rest in bed; so that results were not influenced by these physiologic causes of variation. Under such conditions the p_H of a person [10] as was repeatedly shown in the prefasting period, varies within a very small range, so that the change in p_H which was found in these cases was presumably the direct effect of the fast.

Throughout this series, it has been the endeavor to exclude the effect of convulsions on the blood findings, and no data were included from specimens taken immediately (less than an hour) before or after convulsions. The necessity of observing this precaution was well illustrated

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7. The color comparisons were made at 20 C., using the correction factor given by Cullen. This factor has recently been criticized, and the statement made that all comparisons should be made at the temperature of the patient. The results are not invalidated, however, because they show the relative variation of p_H in a given case.

8. Cullen, G. E.: Studies of Acidosis: Colorimetric Determinations of Hydrogen-Ion Concentration of Blood Plasma, J. Biol. Chem. **52**:501 (June) 1922.

9. Barr, D. P.; Himwich, H. E., and Green, R. P.: Studies in the Physiology of Muscular Exercise; Changes in Acid-Base Equilibrium Following Short Periods of Vigorous Muscular Exercise, J. Biol. Chem. **55**:495 (March) 1923.

10. Eleven control p_H determinations (Henderson-Hassalbach formula) on J. B. gave very consistent results, with an average p_H of 7.36 and the greatest variation 0.03.

TABLE 5.—*p_H of the Plasma*

Case	Control before		Days of Fast													Control after	
			1	2	3	4	5	6	7	8	9	10	11	12	13	14	
1. J. B. (H. H. formula)	....	7.40	....	....	7.30	7.33	7.32	....	7.28	....	....	....	....	7.39	No fast		7.44	....
4. H. A. (Cullen)	....	7.41	7.42	7.41	7.40	7.42	7.32	7.33	7.36	7.35	7.39	7.41		Fast ended			7.45	....
5. P. G. (Cullen)	7.51	7.45	7.44	7.40	7.30	7.31	7.32	7.34	7.35	7.40	7.39	7.40		Fast ended			7.41	7.40

TABLE 6.—*Minute Volume of Respiration (Liters)*

Case	Control before		Days of Fast										Control after		
			1	2	3	4	5	6	7	8	9	10			
4. H. A.	....	4.78	3.74	4.60	4.66	4.86	4.75	5.58	5.05	5.57	5.48	....	4.58	5.15	4.95
5. P. G.	4.61	5.57	6.82	5.68	5.27	5.58	5.94	5.87	4.57	4.82	5.08	4.47	4.74	4.31	....

in one of our cases. Sixty hours after the beginning of a fast, blood was secured from P. G. (Case 5) at the end of a violent convulsion while the child was still unconscious. This was shown to have a whole blood carbon dioxid content of 22 per cent. by volume, and a p_H of 7.07. Her normal p_H was 7.44. This is a profound change and illustrates the effect of a convulsion, a violet form of muscular exercise, on the p_H. Doubtless this change was brought about by two factors, the effect of a convulsion and also of fasting. Three hours later, when the effects of the convulsion presumably had disappeared, the p_H was 7.30 and the whole blood carbon dioxid content was 31 per cent. by volume. Furthermore, the next morning, with no intervening convulsions, the p_H was 7.31 and the carbon dioxid content was 34 per cent. by volume.

Formerly it was believed that changes in the p_H did not occur with ease and that a definite shift was found only under extremely abnormal conditions. The older view was that in "acidosis" a change occurred only in the alkali reserve and that any tendency toward a shift in the p_H was compensated for by means of the delicate interrelation of the blood buffers and the respiratory center; that increased pulmonary ventilation brought about the maintenance of a constant p_H by increased elimination of carbon dioxid. This recently observed shift in p_H to the acid side during fasting and other conditions indicates that there is less of an interdependence of the blood reaction and the respiratory exchange than was formerly thought.[11]

It has been noted in all fasts conducted here that no clinically noticeable increase in respiratory rate has accompanied the acidosis. It was supposed that an increased volume of respiration might be present as a result of the effect of the acidosis on the respiratory center. In Cases 4 and 5 determinations of the minute volume of respiration were made by the spirograph.

During respiratory observations, extreme care was observed to have the child absolutely at rest and free from external stimuli, and several determinations were made each day. The same difficulty is experienced here as was encountered in procuring samples of alveolar air, namely, that of securing adequate cooperation from the child. Determinations by this method are subject to at least 10 per cent. of experimental error.

With these facts in mind, it can only be said that no striking increase in the minute volume of respiration accompanied the acidosis at any stage and that changes in the respiratory volume to a degree even approaching that necessary for compensation of the p_H did not demonstrably develop in these cases. Although the respiration was not shown to have compensated for the change in p_H, it is shown in Table 5

11. MacLeod, J. J. R.: Physiology and Biochemistry, St. Louis, C. V. Mosby Company, 1920. Haldane, J. S.: Respiration, Connecticut, Yale University Press, 1922.

that after a minimum p_H was reached it increased steadily to nearly normal levels, and it is probable that some metabolic change other than the respiration was responsible for this compensation.

Throughout this series, it can be noted that the maximum increase in acid production occurs during the early part of the fast as shown by the acetone, the alkali reserve and the p_H of the plasma. As the fast progressed, compensatory changes tended to bring all of these to a more normal status. Just what factors are responsible for this change have not definitely been proved. The change may be brought about by alterations in excretion, by changes in the type of combustion or by other more obscure changes in metabolism. It is possible that, as will be shown later, an increase in the available carbohydrate may possibly account for this readjustment.

Blood sugar concentrations in these cases were followed with particular interest because of the occurrence of markedly low blood sugar in three fasts previously reported.[1] Determinations were made daily by the method of Folin-Wu,[12] and care was taken to precipitate the blood immediately (within from five to fifteen minutes) so that glycosis could not occur. Low values were read against a 100 mg. standard set at 10, and finally against a 1-1 dilution of the 100 mg. standard.

Prior to fasting, all of these children had normal blood sugar concentrations. During the early days of fasting more or less coincident with the fall in alkali reserve, the increase in acetone production, and the increase in hydrogen-ion concentration, a steady diminution in the blood sugar concentration took place. The minimum was reached between the third and eighth day of fasting and values from 38 to 49 mg. were found with an average minimum of 46 mg. per hundred cubic centimeters. On subsequent days there was a general tendency of the sugar concentration to rise, so that toward the end of the fast values were found very close to the normal range.

It has been repeatedly stated that the blood sugar is not much diminished during fasting. Allen is quoted as saying that the blood sugar is maintained at the normal level (from 80 to 100 mg.) throughout prolonged starvation nearly up to death. Cannon and others [13] state that the blood sugar is persistently maintained at this physiologic level.

Joslin [14] reports the finding of a normal blood sugar concentration throughout the course of a single short fast in a normal adult.

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12. Folin, O., and Wu, H.: System of Blood Analysis: Improved Method for Sugar Determination, J. Biol. Chem. **41**:367 (March) 1920.

13. Cannon, W. B.; McIver, M., and Bliss, S. W.: Effect of Blood Sugar Level on Adrenal Secretion and Sympathetic Activity, Boston M. & S. J. **189**: 141 (July 26) 1923.

14. Joslin, E. P.: Diabetic Metabolism with High and Low Diets, Carnegie Inst., Washington, Publication **323**:137, 1923.

TABLE 7.—*Blood Sugar Concentrations. Milligrams per Hundred Cubic Centimeters*

Case	Control before			Days of Fast													Control after			
				1	2	3	4	5	6	7	8	9	10	11	12	13	14				
1. J. B.	75	74	83	..	..	38	58	55	..	53	..	..	58	..	58	Fast ended		..	81	..	108
2. W. P.	..	..	92	..	73	89	55	70	62	61	49	61	66	64	77	58	74	87	..	76	..
3. C. P.	83	..	92	..	82	68	68	..	68	59	48	57	76	56	70	68	64	95	..	85	..
4. H. A.	101	109	101	94	87	63	68	71	48	61	60	67	78		Fast ended			115	..	..	..
5. P.G.	..	91	..	87	73	76	74	60	46	67	50	60	53		Fast ended			62	90	..	74

TABLE 8.—*Respiratory Quotients*

Case	Control		Days of Fast									
			1	2	3	4	5	6	7	8	9	10
4. H. A.	0.87	0.85	0.78	0.81	0.75	....	0.77	....	0.69	0.70	0.67	....
5. P. G.	....	....	0.79	....	....	0.70	....	0.70	....	0.71	....	....

Weeks and others [15] have reported a series of adult epileptic patients fasted for twenty-one days, on whom weekly blood sugar determinations were made. During starvation, they found in general an increase in blood sugar but it is a question if this rise is not partially accounted for by the interference of acetone with the Benedict method of determination.[16] Two blood sugar determinations in their series were found below 60 mg., but both of these were at the beginning of the fasting period.

Lennox has found, during fasts of varying length on six epileptic and two normal adults, that the sugar level was for the most part well within the normal range, and he found no values at all comparable to the present series.

Since in fasting adults, both normal and epileptic, no diminution in the blood sugar concentration has been demonstrated, it seems fair to assume that the hypoglycemia in our fasting cases was not the result of the epileptic tendency, but represented a change peculiar to childhood. This difference in early life is possibly of some significance.

As this is being written we note a report of work done coincidently by Rumpf,[17] in which he describes the occurrence of blood sugar concentration as low as 45 mg. in normal infants at the end of fasts of twenty-four hours duration. He alludes to studies of Moglitz, who found a similarly marked drop in the blood sugar of infants during fasts of from forty-eight to seventy-two hours. In children past infancy, Rumpf found a moderately marked decrease in the blood sugar, and states that the younger the child and the more prolonged the fast, the more pronounced was the fall in the sugar level.

In childhood when food is withheld the blood sugar concentration diminishes very markedly during the first few days. This might conceivably point to some disturbance inhibiting mobilization of sugar, or to such extremely rapid utilization of the mobilized glucose that the carbohydrate stores are unable to maintain the blood sugar at normal levels. It seems far more likely, however, that the low blood sugar concentrations point to an extreme depletion of the glycogen reserves, and this accords with the fact that in fasting animals a rapid decrease in the glycogen reserves has been demonstrated. The type of combustion occurring in these cases is indicated in the last two patients in Table 8, on whom respiratory quotients were obtained.

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15. Weeks, D. F., et al.: Fasting and Diets in the Treatment of Epilepsy, J. Metabolic Res. **3**:317 (Feb.) 1923.

16. Cowie, D. M., and Parson, J. P.: Studies on Blood Sugar: Effects of Blood Constituents on Picrate Solutions: A Consideration of Limit of the Modified Lewis Benedict Test, Arch. Int. Med. **26**:333 (Sept.) 1920.

17. Rumpf: Uber den Blutsucker im Hunger, etc., Jahrb. f. Kinderh. **105**:317 (March) 1924.

In Cases 4 and 5, H. A. and P. G., frequent determinations of the respiratory quotient were made before and during the fast, using the Benedict cot chamber apparatus. Comparison of Table 7 and Table 8 shows that at about the time minimum sugar levels were reached, the quotient had fallen to approximately that of pure fat combustion, indicating further the reduction of the glycogen reserves.

During the period in which marked hypoglycemia is found, there is a rapid development of acidosis. This points again to depletion of the carbohydrate stores, since with diminished carbohydrate the combustion fat, which then becomes the chief source of energy, is incompletely burned and the acid products of incomplete fat combustion accumulate.

After reaching a minimum the blood sugar slowly rises, sometimes reaching almost normal levels. If glycogen reserves are exhausted early in the fast this later rise is necessarily explained by glucose synthesis. The respiratory quotients during this period are not incompatible with this view. This agrees with the well established fact that in starvation both in adults and experimental animals glucose is synthesized from protein and perhaps even from fats. During this period of increasing blood sugar, there is an associated diminution in the degree of acidosis. During the later stages of the fasts when the blood sugar has risen, doubtless because of increased glucose synthesis, the acidosis is possibly compensated to some degree by a greater availability of carbohydrate.

These fasts would seem to justify the hypothesis that more rapid and profound exhaustion of the glycogen reserves occur in fasting children than is the case in adults, and that the body is unable immediately to bring about a degree of compensation, by glucose synthesis, sufficient to maintain a normal blood sugar concentration.[18] This would help to account for the fact, long recognized clinically, that a far greater tendency to acidosis is encountered in childhood and it should be pointed out that acute infections, cyclic vomiting and similar phenomena in childhood associated with anorexia and vomiting virtually amount to involuntary fasts.

The subject of low blood sugar concentrations has received a great deal of attention in connection with insulin therapy. The sugar values in this series are well below the level where with insulin administration the so-called hypoglycemic symptoms commonly supervene. Severe hypoglycemic reactions, however, did not occur in any of these patients and in no patient were there symptoms unmistakably due to the low sugar, since moderately marked acidosis was always simultaneously present. In one case, J. B., with a blood sugar concentration of 38 mg. and a plasma combining power of 34 per cent. by volume, the patient

18. Talbot, F. B.; Shaw, E. B., and Moriarty, Margaret E.: Hypoglycemia and Acidosis, J. A. M. A. **83**:91 (July 12) 1924.

complained of feeling very ill and afraid, and of a feeling of oppression and some pain in the chest and throat. She was restless and appeared quite ill. She vomited several times. Clinically the symptoms did not suggest acidosis; there was only a slight flush and no hypernea was present. These symptoms were so disturbing that the patient was given 2 ounces of diluted orange juice with added lactose. This she promptly vomited. Later several teaspoonful doses of the same mixture were given which she retained for about an hour and then vomited. The severe symptoms lasted for about an hour and then subsided. Doubtless some of the sugar administered was absorbed, for twelve hours later the blood sugar concentration had risen to 58 mg. and there was a rise in the alkali reserve and p_H. Through the remainder of the fast she had no similar symptoms and complained very little of discomfort. Similar but much milder symptoms were observed in W. P. on the fourth day of his second fast when the blood sugar was 54 mg. The other patients showed no comparable symptoms and were relatively comfortable throughout their fasts.

It is perhaps significant that in the two instances in which such symptoms were observed there had been a more abrupt drop in the sugar than at any other time.

MacLeod [19] has recently shown that when insulin is given to fasting animals, the blood sugar may be reduced much below the usual threshold of hypoglycemic symptoms without the production of such symptoms.

CONCLUSIONS

It is shown in a series of five epileptic children that from ten to fourteen day fasts were accompanied by:

1. An increase in blood uric acid.
2. Moderately marked acidosis, as indicated by (*a*) acetone excretion in the breath; (*b*) diminished alkali reserve; (*c*) moderate fall in p_H of the plasma.
3. A striking decrease in the blood sugar.
4. A slight diminution in degree of acidosis and rise in the blood sugar during latter days of fast.

It is suggested that the diminution of blood sugar indicates the greater case of depletion of carbohydrate stores in childhood, and helps to explain the increased tendency to acidosis.

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19. MacLeod, J. J. R.: Insulin, Physiol. Rev. **4**:39 (Jan.) 1924.

NONRACHITIC SOFTENING OF THE RIBS IN INFANTS AND CHILDREN *

ALFRED F. HESS, M.D.

NEW YORK

For the last seven years the routine physical examination of children in this institution has included a special survey for rickets. These examinations have been carried out almost every month on infants under 18 months of age and the data recorded on separate forms. In addition to noting the presence or absence of purely clinical signs, a radiograph of the epiphyses of the wrist has been included for the last five years, and an estimation of the inorganic phosphate of the blood for the last three years. In the course of these routine tests our attention has frequently been directed to the great variability in the resistance or pliability of the chest wall. When downward pressure is exerted on the anterior thoracic wall, the thumbs being placed on the manubrium of the sternum, there is a greater or less degree of yielding of the ribs. In some cases the costal cartilages and adjacent ends of the ribs offer surprisingly little resistance to such pressure, the yielding being most pronounced at their junctions. The test should be carried out preferably during expiration, and cannot satisfactorily be elicited while the child is taking an inspiration. This varying flexibility of the chest wall attracted our attention about five years ago, and since this time has been recorded in many cases in connection with the routine tests for rickets. The circumstance that most of the babies remain in the institution until they are about 5 years of age has afforded an exceptional opportunity for observing the progress of this phenomenon, and of noting its response to such therapeutic measures as are of specific value in rickets.

This lack of resistance of the ribs has been noted frequently and commented on by others and is generally accepted as characteristic of rickets. Kassowitz,[1] one of the keenest students of rickets, calls attention to it, and Siegert [2] took the position that the presence of "abnormal softening of the ribs" establishes the diagnosis of rickets. Only lately, Moore [3] emphasized its significance in discussing the newer signs of rickets.

As is true of all subjective signs, for example beading of the ribs, it is not possible to draw a sharp boundary line between the normal and

* Received for publication, Aug. 12, 1924.

* From the Home for Hebrew Infants.

1. Kassowitz, M.: Praktische, Kinderheilkunde, Berlin, J. Springer, 1914.

2. Siegert, F.: Jahrb. f. Kinderh. **58**:929, 1903.

3. Moore, C. U.: Newer Clinical Signs of Early Rickets, J. A. M. A., to be published.

the pathologic. This is especially relevant to softening of the ribs. The difference, however, between a normal and a highly compressible chest wall is unmistakable. As the basis for this study only extreme deviations have been considered, all questionable or borderline cases having been disregarded. The difficulty in determining whether a particular symptom is of rachitic origin has always been that, although there may be no signs whatsoever of rickets at the time of examination, the possibility always exists that signs may subsequently develop. This situation is complicated further by the fact that almost all bottle fed infants develop rickets unless special precautions are taken; this is true if the newer and more delicate diagnostic tests are made use of and if the examinations are conducted in the Spring. There is danger, therefore, that the association with rickets of any phenomenon may be merely fortuitous.

To obviate this difficulty the therapeutic test was used, the effect of specific antirachitic treatment. This was carried out by giving cod liver oil, yolk of egg or a combination of both, or irradiating with sunlight or with the carbon arc light. In addition to these measures which are specifically curative for rickets, autolyzed yeast was given to determine whether a lack of the water-soluble vitamin could be at fault, iron was tried in the form of the saccharated carbonate, and calcium as calcium lactate. Ten infants were given cod liver oil, seven infants egg yolk, and five infants one or other form of ultraviolet irradiation. In some instances this treatment was carried out for a year; this was especially the case with cod liver oil or with the yolk of egg; in no instance was the treatment given for less than three months. The following is an abstract from the records of ten typical cases, which have been seleeted to illustrate the various therapeutic procedures.

ABSTRACT OF CASES

CASE 1.—A. A., a girl, was admitted when 1 month of age, weighing 6 pounds (2.7 kg.). She has had marasmus, and at the age of 5½ years weighed 33½ pounds (15 kg.). The child was mentally backward. The head was large, and she had moderate rickets. The ribs remained soft throughout this period, in spite of the yolk of an egg having been given daily for 4½ months, and three teaspoonfuls of cod liver oil from the age of 7 to 14½ months.

CASE 2.—E. W., a girl, was admitted when 5 months of age, weighing 10¾ pounds (4.5 kg.). When 4 years old she weighed 31 pounds (14 kg.). She appeared to be a poorly nourished child and had moderate rickets. When 15 months of age she sat up, and had her first tooth at the age of 13 months. The ribs were soft; this condition was first noted when aged 19 months. The circumference of the head was 4 cm. larger than that of the chest. The chest was flattened. The child received 6 teaspoonfuls daily of cod liver oil for over four months and cabbage at the age of 11 months. The child received no benefit from this treatment.

CASE 3.—O. S., a boy, was admitted when aged 5 months, weighing 13 pounds, 2 ounces (5.9 kg.). At 1 year he weighed 14½ pounds (6.5 kg.), at 20 months

he weighed 22 pounds, 7 ounces (10.4 kg.). He was a poorly nourished baby who had been fed on raw certified milk. He had slight rickets; the inorganic blood phosphorus tests were 3.52 mg., 3.44 mg. and 4 mg. Six roentgenograms were negative. Moderate "rosary," slight Harrison's groove. The child stood at the age of 14 months. The ribs were noted to be soft from the date of admission.

CASE 4.—C. K., a girl, was admitted when 6 weeks old, weighing 8 pounds, 6 ounces (3.6 kg.). At 1½ years she weighed 21 pounds, 13 ounces (14.1 kg.) and had eight teeth. She stood at the age of 11½ months. Ten roentgenograms were negative. Five blood tests ranged from 4 to 4.6 mg. of inorganic phosphorus, calcium 10.8 mg. Slight "beading" was noted at the age of 3 months. Egg yolk was given for seven months. The soft ribs persisted.

CASE 5.—M. S., a boy, twin, was admitted when 1 month old, weighing 5 pounds, 15 ounces (2.7 kg.). At 2 years of age he weighed 22½ pounds (10 kg.). The child had moderate rickets which condition was cured by egg yolk which was given for four months. The inorganic phosphorus rose to normal and roentgenograms became negative. The bones became strong. The head was large and the mentality questionable. The soft ribs persisted.

CASE 6.—M. L., a boy, was admitted when 5 weeks of age, weighing 6¾ pounds (3.2 kg.), and poorly nourished. At 20 months the child weighed over 26 pounds (11.8 kg.). Moderate rickets was noted, but under treatment the blood phosphorus rose to 4 mg. at the age of 14 months. The fontanel closed at the age of 17 months; the head was large for the first six months of life. The child stood at 14 and walked at 18 months. Malocclusion of the jaws was present. The patient was treated with sunlight from July to October. The rickets was cured, but the sunlight had no effect on the ribs.

CASE 7.—F. F., a boy, was admitted at the age of 3 months, weighing 7 pounds, 15 ounces (3.6 kg.). Marasmus was present. At 2⅓ years old he weighed 30 pounds (13.6 kg.). He was a fine child at 1 year. Moderate rickets was observed both clinically and by roentgenogram. The inorganic phosphorus was 3.57 mg. The teeth were discolored. The child was treated with carbon arc rays. The rickets was cured but the soft ribs persisted.

CASE 8.—C. B., a boy, was admitted when 3 weeks old, weighing 6 pounds (2.7 kg.). He was a poorly nourished child. At 2 years he weighed 24½ pounds (10.9 kg.). Slight rickets was cured with carbon arc rays, so that the inorganic phosphate rose to 4.27 mg. at the age of 13 months. The child had been fed on certified raw milk and was anemic. Iron treatment was given. His mentality was backward, and the child was given thyroid extract. The soft ribs were noted at the ages of 7, 8, 12, 13 and 20 months. Carbon arc treatment did not harden ribs ("chicken-breast").

CASE 9.—I. A., a boy, was admitted when 3 months of age, weighing 10 pounds, 6 ounces (4.5 kg.), and was poorly nourished. At 7½ months he weighed only 13½ pounds (5.9 kg.). At the age of 3¼ years he weighed 32¼ pounds (14.5 kg.). Slight rickets was shown on the tenth roentgenogram. The head was large. Up to the age of 15 months the child did not sit up. The teeth were small and poorly formed. "Chicken-breast." The patient was given autolyzed yeast for six months without benefiting the ribs.

CASE 10.—J. S., a boy, was admitted when 9 months old, weighing 14 pounds, 5 ounces (6.4 kg.), and was poorly nourished. There were no signs of rickets. The inorganic phosphorus was 4.4 mg., calcium 10.6 mg. The fontanel closed at the age of 18 months. The child had thirteen teeth. He was given iron (saccharated carbonate, 5 grains daily) for a year but the soft ribs persisted.

COMMENT

Discussion of our results should be prefaced by the statement that it is the general experience that all symptoms and signs of rickets, whatever their nature, clinical, radiologic or chemical, are alleviated or cured by cod liver oil or ultraviolet irradiation. It is significant therefore, that neither of these forms of specific treatment lessened the softening of the ribs. It may be added that seasonal variation, which plays such a dominant rôle in the development and cure of rickets, was found to be of no moment in relation to the appearance or recession of this sign, no improvement whatsoever taking place during the summer months. In other words, in marked contrast to rickets, this condition is not amenable to treatment.[4]

A striking feature is that the softening was found almost always in infants who were or had been poorly nourished. This characteristic will be noted throughout the records of the ten illustrative cases; none of these were fine, healthy babies. This is not a characteristic of rickets. As is well known, rickets is frequently met with in babies who are overweight and robust. In this type of baby, however, rickets is not associated with softening of the ribs. For example: A healthy child, aged 3 years, who has always been robust. He has had rickets and still has roentgenologic signs of an active process. There is a marked "rosary," very marked bowing of the legs, moderate enlargement of the epiphyses, and by roentgen-ray examination moderate rickets with healing. In spite of this long period of rickets, noted incidentally in the course of this survey, the chest wall is especially firm. This distinction is one which should be stressed in considering the relation of softening of the ribs to rickets. Although rickets tends, no doubt, to emphasize its intensity, it has a closer relationship to malnutrition and atrophy. For example, it developed in marked degree in an infant which acquired miliary tuberculosis. Under these conditions, attended by progressive loss of weight, rickets has no tendency whatsoever to develop. In this case the softening was corroborated at necropsy, and the costochondral junctions were found to be the site of osteoporosis and not of rickets. It is quite possible that histologic examination would frequently reveal osteoporosis. In the course of inducing rickets in rats, osteoporosis is often noted in animals which do not show any lesions of rickets; these animals are almost always poorly nourished.

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4. Instead of giving cod liver oil we have substituted this year phosphorus in almond oil, according to the formula of Kassowitz. In doing this we had in mind the possibility that the success of Kassowitz with phosphorus might be due in part to the sclerotic action of phosphorus in cases with softening of the ribs. It is too early to express an opinion as to its value.

An examination of newly-born infants emphasizes this relationship. A large number of babies, white as well as colored, were examined at the Sloane Hospital for Women, the physicians of which I take this opportunity to thank for their cooperation. In this connection it may be well to mention again the difficulty in differentiating between degrees of flexibility of the chest wall in infants. No one, however, can fail to be struck by the abnormal compressibility of the thorax in premature infants. Almost all premature infants have soft ribs; only one baby, weighing less than 5 pounds, was found to have a firm thoracic wall. On the other hand the instances of large babies having compressible chests were exceptional. This softness of the ribs of the new-born cannot be considered a manifestation of rickets unless we are to believe that this group is suffering from congenital rickets. In a previous paper [5] it was reported that the blood of premature infants contained a normal percentage of inorganic phosphorus and calcium. It may be added that roentgen-ray examination of twenty-five premature infants has given no evidence of rickets. The lack of firmness in these infants frequently persists for years. Among the children in our institution, about 375 in all, there are thirty-four with soft ribs and ten of this number were born prematurely.

As stated, it is difficult to study infants which, we are certain, never had rickets. In order to be sure regarding this point, they must have been tested by the newer methods, as well as have been under observation for a period sufficiently long and continuous to exclude all possibility of this disorder. We have had opportunity of observing for a period of two years or more, several infants which have shown none of the signs of rickets. Softness of the ribs, however, was frequently noted. As an example the following instance is given.

B. B., a boy, was admitted February, 1921, when 4 months of age. He was poorly nourished, weighing 9 pounds, 7 ounces (4.1 kg.). At the age of 6 months he weighed 11½ pounds, and 15¾ pounds at a year. During this period he showed no signs of rickets; no bleeding of the ribs, enlarged epiphyses, craniotabes, etc. He had his first tooth when aged 7 months, and he stood at 18 months. Seven roentgenograms were negative during the first year and nine others during the course of the next year. The inorganic blood phosphate was estimated seven times at intervals, and varied between 3.89 mg. and 4.54 mg. per cent. The diet consisted of simple dilutions of milk, of cereal and orange juice. In spite of the absence of all rachitic signs, the ribs were persistently soft.

No association was noted between this clinical condition and any particular sign of rickets. It was not associated, for example, more often with craniotabes than with beading of the ribs or enlarged epiphyses. The only peculiarity was that the circumference of the heads

5. Hess, Alfred F., and Matzner, Milton J.: Rickets in Relation to the Inorganic Phosphate and Calcium in Maternal and Fetal Blood, Am. J. Dis. Child. **26**:285 (Sept.) 1923.

of many of these infants was large in comparison to that of their chests; this tended to give them a rachitic appearance. Some infants seemed to have a slight hydrocephalus accompanied by a widening of the fontanel. The softening of the ribs and their cartilages led to a sinking of the anterior wall of the thorax, to a flattening of the chest. This change in conformation was apparent in some instances on casual observation and was confirmed by measurements of the thorax. Although the circumference of the chest was normal in infants with soft ribs, the anteroposterior diameter was less in comparison to the lateral diameter than in infants with firm ribs. This frequently resulted in a permanently flattened chest. Some of the children had the deformity of the chest, commonly termed "chicken-breast" or a "funnel-shaped chest." These deformities may result from rickets but they may likewise come about from other causes. Pneumonia may lead to "chicken-breast"; this occurred in a twin who had a soft chest but no rickets, and who had had recurrent pneumonia. The most common etiologic factor of these two malformations is rickets combined with softness of the ribs. Among the case histories which we have given in detail are included two instances of "chicken-breast" and one of "funnel-shaped chest." Both the "chicken-breast" and "funnel-shaped" chest were observed in new-born infants. In one instance with "funnel-shaped chest" the baby weighed over 9 pounds; the mother's chest was normal.

CONCLUSION

Softness of the ribs and of the anterior thoracic wall is not necessarily a sign of rickets, although it may be brought about or intensified by this disorder. It is associated preeminently with the atrophic in contradistinction to the well nourished infant, two groups which should be differentiated in considering rickets. Infants who have never had rickets may nevertheless show softening of the ribs. It is found frequently in the new-born, especially those born prematurely, and may persist for years. In these cases there can be no question of a rachitic origin. Finally, it is not alleviated or cured by prolonged treatment with the two specifics for rickets, cod liver oil or ultraviolet irradiation.

THE RELATION OF CALCIUM AND PHOSPHORUS IN THE DIET TO THE ABSORPTION OF THESE ELEMENTS FROM THE INTESTINE *

W. J. ORR, M.D.; L. E. HOLT, JR., M.D.; L. WILKINS, M.D.
AND
F. H. BOONE, M.D.
BALTIMORE

The problem of absorption from the intestine has taken on a new significance since it has been appreciated that the failure of calcification in rickets is coincident with and apparently dependent on the absorption of minerals. The experiments of Schabad,[1] Schloss [2] and others, including us,[3] have shown that active rickets is associated with defective absorption of calcium and phosphorus, and that as the disease heals, large quantities of these elements are retained. Moreover, it seems clear that the absorption of these elements can be increased in at least two ways: (1) by the administration of a factor contained in certain animal fats, of which cod liver oil is the best example; and (2) by the use of ultraviolet rays. Both of these measures are effective, even though the quantity of calcium and phosphorus supplied varies greatly. It has been supposed that these substances act by altering in some way the permeability of the intestinal wall toward these particular elements.

Although cod liver oil and ultraviolet rays are the methods by which the absorption of these elements has been effected experimentally, still there are other factors which may presumably influence the process, such as the concentration of calcium and phosphorus supplied in the diet, the reaction of the intestinal contents, variations in the concentration of other salts, changes in temperature, etc. It would seem that considerable light

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* Received for publication, July 29, 1924.

* From the Department of Pediatrics, Johns Hopkins University, and the Harriet Lane Home, Johns Hopkins Hospital.

1. Schabad, J. A.: Ztschr. f. klin. Med. **66**:454, 1908-1909; ibid. **68**:94, 1909; ibid. **69**:435, 1910; Berl. klin. Wchnschr. **46**:823, 1909; ibid. **46**:923, 1909; Jahrb. f. Kinderh. **72**:1, 1910; ibid. **74**:511, 1911; Arch. f. Kinderh. **52**:47-68, 1909; ibid. **53**:380, 1910; ibid. **54**:83, 1910; Ztschr. f. Kinderh. **2**:117, 1911; Monatschr. f. Kinderh. **11**:63, 1912. Schabad and Soroschowitch: Monatschr. f. Kinderh. **9**:659, 1911; ibid. **10**:12, 1912; Arch. f. Kinderh. **57**:276, 1912.

2. Schloss, E.: Deutsch. med. Wchnschr. **39**:1505, 1913; Arch. f. Kinderh. **63**:359, 1914; Jahrb. f. Kinderh. **78**:694, 1913; ibid. **79**:40, 194, 1914; ibid. **82**:435, 1915; ibid. **83**:46, 1916. Frank and Schloss: Jahrb. f. Kinderh. **79**:539, 1914; Biochem. Ztschr. **60**:378, 1914; Monatschr. f. Kinderh. **13**:271, 1914.

3. Orr, W. J.; Holt, L. E.; Wilkins, L., and Boone, F. H.: The Calcium and Phosphorus Metabolism in Rickets, with Special Reference to Ultraviolet Therapy, Am. J. Dis. Child. **26**:362 (July) 1923.

might be thrown on the process of absorption in general by an investigation of these various factors separately. In this instance we have studied only the effect of varying the concentrations of calcium and phosphorus in the diet on the absorption of these elements.

Considerable work has already been done on this subject but much of it is open to criticism, either because the variation of the elements in the diet was so small that little significance could be attached to the results, or because the effect of other factors, such as cod liver oil and light rays, were not excluded.

Largely as a result of the earlier work [4] on the subject, the conclusion has been accepted that feeding an excess of one element impairs the absorption of the other, although an increased amount of the element fed in excess may be retained. There is, however, contradictory evidence [5] on this point. Metabolism experiments have been the means employed to study the question. In more recent years studies of the blood have furnished additional information in regard to the absorption of these particular elements, a rise of concentration in the serum being taken as an indication of increased absorption, and a fall, of impaired absorption.

Data are available in regard to the alterations in the concentrations of calcium and phosphorus in the blood, produced by varying the intake of these elements. Boggs [6] reports an increase in blood calcium in dogs after feeding calcium lactate. Meigs, Blatherwick and Cary [7] report experiments with cattle, in which calcium chlorid or disodium phosphate was added to the diet. Excessive calcium in the diet caused an increase in the plasma calcium but a diminution of the plasma phosphorus; while, on the other hand, excessive phosphate in the diet raised the phosphorus concentration of the plasma.

The production of experimental rickets in rats by McCollum, Simmonds, Shipley and Park [8] on a diet high in calcium but low in phosphorus has furnished more evidence that calcium absorption is thus improved and phosphorus absorption impaired, for Howland and Kramer [9] have shown that in these animals the calcium concentration of the blood serum is raised and that of the inorganic phosphorus

4. Bertram, J.: Ztschr. f. Biol. **14**:335, 1878. Hammarsten: Text Book of Physiologic Chemistry, Ed. 7, 1915, 762. Forbes, E. B., et al.: Ohio Agric. Exp. Sta., Tech. Bull. **6**:66, 1915. Meyer and Cohn: Quoted by Blühdorn, Footnote 15.

5. Quoted by Blühdorn, Footnote 15.

6. Boggs, T. R.: Bull. Johns Hopkins Hosp. **19**:201, 1908.

7. Meigs, E. B.; Blatherwick, N. R., and Cary, C. A.: J. Biol. Chem. **37**: 63, 1919.

8. McCollum, E. V.; Simmonds, N.; Shipley, P. G., and Park, E. A.: J. Biol. Chem. **47**:507 (Aug.) 1921.

9. Howland, J., and Kramer, B.: Bull. Johns Hopkins Hosp. **33**:313 (Sept.) 1922.

lowered. Furthermore, when rickets is produced by a diet low in calcium and relatively high in phosphorus,[10] the concentration of serum phosphorus is increased and that of the calcium diminished.[9]

Thus all of the reported experiments[11] on animals confirm the original suggestion that an excess either of calcium or phosphorus in the diet exercises an unfavorable influence on the absorption of the other element from the intestine. The natural assumption that has been made to explain this is, that an excess of one element in the diet causes precipitation of the other element in the intestine in the form of insoluble calcium phosphate, which is eliminated in the feces.

We have been able to find records of very few experiments in man which are free from error or at least criticism. Schabad[12] reported observations on a child to whom he gave an excess of sodium phosphate, with resulting improvement in the phosphorus retention, but with definite impairment of the calcium balance. He also tried the effect of feeding calcium acetate, with the result that an excessive loss of phosphorus occurred in the feces. Bosworth and Bowditch[13] performed a somewhat similar experiment. They gave a child an excess of calcium chlorid and found an increased retention of calcium, but a marked loss of phosphorus in the feces.[14]

The work of Blühdorn,[15] however, would suggest an entirely different conclusion. He studied the metabolism of two children who received a normal diet, diets with an excess of calcium and those with an excess of phosphorus. He concluded from his experiments that calcium and phosphorus absorption run parallel. An excess of either element in the diet caused an increase in the quantity of both retained. He found no evidence for an unfavorable effect of excessive calcium on the phosphorus metabolism, or of excessive phosphorus on the calcium metabolism.

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10. McCollum, E. V.; Simmonds, N., and Kinney, E. M.: Am. J. Hygiene **2**:97 (March) 1922.

11. Since this was written, the experiments of Salvesen, Hastings and McIntosh on dogs have been published (J. Biol. Chem. **60**:311-327 [June] 1922). They, too, find that oral administration of phosphates cause a rise of inorganic phosphorus in the serum, and a drop in the serum calcium. Their experiments with oral administration of calcium salts, however, did not give such striking results.

12. Schabad, J. A.: Arch. f. Kinderh. **54**:83, 1910.

13. Bosworth, A. W., and Bowditch, H. I.: Boston M. & S. J. **177**:864 (Dec. 20) 1917.

14. In this connection the studies of Howland and Kramer on tetany are of interest (Tr. Am. Ped. Soc., 1922, p. 204). These observers found that feeding calcium raised the concentration of the calcium in the blood serum, but caused a coincident fall in the phosphorus content of the serum. The mineral metabolism of their patients was not studied, but the results were nevertheless highly suggestive.

15. Blühdorn, K.: Ztschr. f. Kinderh. **29**:43 (April) 1921.

These results of Blühdorn seemed so surprising and so much in conflict with animal experiments and the few other uncomplicated experiments with children, that we undertook to investigate this point further,

TABLE 1.—*Experiments on Child with Rickets*

B. G., colored child, aged 18 months, clinical signs of rickets. Admitted March 26, 1923.

Serum determinations, 3/26/23: Ca = 10.2 mg./100 c.c.; P = 2.8 mg./100 c.c.

First Metabolism Period: Four days (4/4/23—4/8/23) normal milk diet.

Ca intake	3.98 gm.	P intake	2.84 gm.
Ca urine output	0.03 gm.	P urine output	1.46 gm.
Ca stool output	2.86 gm.	P stool output	0.72 gm.
Total Ca output	2.89 gm.	Total P output	2.18 gm.
Ca retention	1.09 gm.	P retention	0.66 gm.
Per cent. retained	27.1	Per cent. retained	23.4
Per cent. output in urine	1.3	Per cent. output in urine	66.9

Ratio Ca/P retained 1.65

Serum determinations, 4/8/23: Ca = 8.4 mg./100 c.c.; P = 4.0 mg./100 c.c.

Second Metabolism Period: Four days (4/16/23—4/20/23) Diet: milk + $CaCl_2$.

Ca intake	9.20 gm.	P intake	2.80 gm.
Ca urine output	0.30 gm.	P urine output	1.22 gm.
Ca stool output	7.12 gm.	P stool output	1.16 gm.
Total Ca output	7.42 gm.	Total P output	2.38 gm.
Ca retention	1.78 gm.	P retention	0.42 gm.
Per cent. retained	19.4	Per cent. P retained	15.1
Per cent. output in urine	4.0	Per cent. P output in urine	51.

Ratio Ca/P retained 4.2

Serum determinations, 4/19/23: Ca = 10.2 mg./100 c.c.; P = 3.1 mg./100 c.c.

Third Metabolism Period: Four days* (4/30/23—5/3/23) Diet: milk + Na_2HPO_4.

Ca intake	3.41 gm.	P intake	5.42 gm.
Ca urine output	0.04 gm.	P uterine output	1.49 gm.
Ca stool output	3.26 gm.	P stool output	3.47 gm.
Total retention	3.30 gm.	Total P output	4.96 gm.
Ca retention	0.11 gm.	P retention	0.46 gm.
Per cent. retained	3.2	Per cent. P retained	8.4
Per cent. output in urine	1.2	Per cent. P output in urine	30.0

Ratio Ca/P retained 0.24

Serum determinations, 5/4/23: Ca = 7.55 mg./100 c.c.; P = 4.1 mg./100 c.c.

Subsequent Course: Antirachitic treatment was started immediately after third metabolism period, with resulting cure of the rickets.

Serum determinations, 5/14/23: Ca = 10.0 mg./100 c.c.; P. = 4.0.

Serum determinations, 5/23/23: Ca = 10.8 mg./100 c.c.; P = 5.0.

Child discharged in good condition 5/24/23.

* Child developed diarrhea on the third day and the experiment was stopped. Results calculated to four-day period (multiplied by ⅓) for the sake of comparison.

in order to determine if the conditions found in the lower animals are applicable to man.

Two subjects were selected for this study: one a child with incipient rickets, the other a normal child who had previously suffered from a

mild nutritional disturbance. The calcium and phosphorus intake and output of these children was determined on three different diets: a normal diet, a high calcium diet, and a high phosphorus diet. Suitable intervals were allowed to elapse between these periods of study. Determinations of the calcium and phosphorus of the serum were made

TABLE 2.—*Experiment on Undernourished Child*

C. S., colored child, aged 14 months, somewhat undernourished. Admitted April 1, 1923.

Serum determinations, 4/4/23: Ca = 10.0 mg./100 c.c.; P = 4.9 mg./100 c.c.

First Metabolism Period: Four days (4/4/23—4/8/23) on normal milk diet.

Ca intake	3.98 gm.	P intake	2.84 gm.
Ca urine output	0.09 gm.	P urine output	1.37 gm.
Ca stool output	2.86 gm.	P stool output	0.68 gm.
Total Ca output	2.95 gm.	Total P output	2.05 gm.
Ca retention	1.03 gm.	P retention	0.79 gm.
Per cent. retained	26.0	Per cent. retained	27.6
Per cent. output in urine	3.1	Per cent. output in urine	66.6

Ratio Ca/P retained 1.30

Serum determinations, 4/16/23: Ca = 9.0 mg./100 c.c.; P = 5.4 mg./100 c.c.

Second Metabolism Period: Four days (4/16/23—4/20/23) Diet: milk + $CaCl_2$.

Ca intake	7.29 gm.	P intake	2.13 gm.
Ca urine output	0.41 gm.	P urine output	1.02 gm.
Ca stool output	4.84 gm.	P stool output	0.94 gm.
Total Ca output	5.25 gm.	Total P output	1.96 gm.
Ca retention	2.04 gm.	P retention	0.17 gm.
Per cent. Ca retained	27.9	Per cent. retained	8.0
Per cent. output in urine	7.7	Per cent. output in urine	52.0

Ratio Ca/P retained 12.0

Serum determinations, 4/21/23: Ca. = 10.0 mg./100 c.c.; P = 3.3 mg./100 c.c.

Third Metabolism Period: Four days (4/30/23—5/4/23) Diet: Milk + Na_2HPO_4.

Ca intake	3.95 gm.	P intake	6.36 gm.
Ca urine output	0.05 gm.	P urine output	2.96 gm.
Ca stool output	3.50 gm.	P stool output	2.82 gm.
Total Ca output	3.55 gm.	Total P output	5.78 gm.
Ca retention	0.40 gm.	P retained	0.58 gm.
Per cent. retained	10.3	Per cent. retained	9.2 gm.
Per cent. output in urine	1.3	Per cent. output in urine	51.0

Ratio Ca/P 0.69

Serum determinations, 5/4/23: Ca = 10.4; P = 5.0
5/7/23: Ca = 10.5; P = 4.3

Discharged 5/8/23 in good condition.

in connection with each period of study. The details of our procedure and the methods used were as reported in a previous publication.[3] Protocols of these experiments are given in Tables 1 and 2.

COMMENT

It is quite evident from these experiments that increasing the intake of calcium causes more calcium to be retained; the serum calcium rises if it is low, and more calcium is lost in the urine. At the same time the

phosphorus retention is definitely diminished, considerable amounts of phosphorus are diverted from the urine to the stools, and there is a diminution of the concentration of phosphorus in the serum.

The effect of high phosphorus intake is almost equally striking. Although the actual amount of phosphorus retained is not increased and may even be somewhat diminished, the concentration of phosphorus in the serum is definitely increased. There is a very marked decrease in the amount of calcium retained, a great increase in the calcium of the stools, a marked diminution of the calcium in the urine, and in one case a definite fall in the concentration of calcium in the serum.

The fact that absorption of calcium and phosphorus does not run parallel is perhaps most clearly demonstrated by examination of the ratio of the retained calcium to the retained phosphorus. It will be seen that on a normal milk diet this ratio is about 1.3, that is, a little more calcium is retained than phosphorus. On the high calcium diet, in one case four times as much calcium as phosphorus is retained, and, in the other case, twelve times as much. On the high phosphorus diet considerably more phosphorus is retained than calcium.

Our results, therefore, seem to contradict the findings of Blühdorn and confirm those of Schabad, of Bowditch and of Bosworth, and the numerous investigators who have worked with lower animals. It would appear that an excess of either calcium or phosphorus in the diet tends to increase the output of the other element in the intestine, and the natural assumption that has been made is that this is due to the precipitation of insoluble phosphates of calcium in the intestine. Apparently only calcium ions, the various types of phosphate ions and molecular phosphates of calcium in solution can pass through the intestinal mucosa, but insoluble phosphates of the calcium cannot be absorbed. If this is true, it should follow that any factor which favors solution of calcium phosphate should promote the absorption of both calcium and phosphorus from the intestine (when they are present in any fixed concentrations). Such factors are: (1) increased acidity of the intestinal contents, with resulting production of the more soluble primary phosphate from the insoluble secondary and tertiary forms; (2) reduction of temperature; (3) increase in the total salt concentration [16] and (4) specific ion effects.

It is, of course, not possible to measure directly the changes in the hydrogen-ion concentration of the intestinal contents, but indirect evi-

16. It has been shown by Brønsted and others that the solubility of sparingly soluble salts is often increased by the presence of foreign salts, and sometimes even by the addition of salts containing a common ion. That this is true of at least one form of insoluble calcium phosphate, the tricalcium phosphate, $Ca_3(PO_4)_2$, has been shown by the unpublished experiments of one of us (L. E. H.).

dence on this point is not wanting. For instance, it has been shown [17] that foods which are acid forming improve calcium retention, while base forming foods have the opposite effect. Moreover Sato [18] noted an unfavorable effect on calcium metabolism when alkali was added to the dietary of an infant. Unfortunately, the phosphorus metabolism was not followed in either of these studies. The experiments of Orr,[19] however, furnish evidence that both phosphorus and calcium absorption are unfavorably influenced by diets that increase the alkalinity of the stools.

The effect of changes in temperature on the absorption of calcium and phosphorus has not been studied, but it would seem that they might exert both a direct and an indirect influence on the process. The solubility of tertiary calcium phosphate is diminished by heat, and one would therefore expect that a rise in temperature would impair absorption. The work of Yllpö [20] suggests that temperature changes may also exert an indirect effect, namely, by altering the reaction of the gastro-intestinal contents. Yllpö has recently shown that fever is often accompanied by decreased acidity in the stomach, and it seems possible that the reaction of the intestinal contents might also be altered.

So far, there have been no investigations of calcium and phosphorus metabolism in which the variable factor has been the total salt concentration.

There is, however, evidence which might be interpreted as indicating that specific ion effects play a part in absorption from the intestine. Goldschmidt and Dayton [21] and Goldschmidt and Binger [22] have shown great differences in the ease with which sodium chlorid is absorbed by the intestine, depending on the presence and concentration of sodium sulphate or calcium lactate. Although these observers interpreted their results as indicating an alteration of the intestinal wall by these latter salts, it seems equally possible, in the light of recent chemical work,[23] that specific interaction of ions might play a part in such phenomena.

We have, then, considerable evidence that absorption of calcium and phosphorus from the intestine depends on the concentration of calcium and phosphate ions, and the presence of soluble undissociated phosphates of calcium. Such physicochemical factors as alter these concentrations of salts or ions or their activities may greatly influence the absorption

17. Bogert, L. J., and Kirkpatrick, E. E.: J. Biol. Chem. **54**:375 (Oct.) 1922.
18. Sato, A.: The Effect of Alkali and Malt Preparations on the Retention of Calcium in Infancy, Am. J. Dis. Child. **16**:293 (Nov.) 1918.
19. Orr, W. J.: Am. J. Dis Child. To be published.
20. Yllpö, A.: Acta Pediat. **3**:213 (Feb.) 1924.
21. Goldschmidt, S., and Dayton, A. B.: Am. J. Physiol. **48**:459 (May) 1919.
22. Goldschmidt, S., and Binger, C. A. L.: Am. J. Physiol. **48**:473 (May) 1919.
23. Brønsted, J. N.: J. Am. Chem. Soc. **42**:761, 1448, 1920; ibid. **43**:2265, 1921; ibid. **44**:877, 938, 1922; ibid. **45**:2898, 1923.

of these elements. The question still remains: how do cod liver oil and ultraviolet light effect the absorption of these elements so profoundly?

SUMMARY

1. It is shown by metabolism studies that excessive amounts of calcium in the diet tend to increase the total absorption and retention of calcium, but tend to impair phosphorus retention.

2. Excessive amounts of phosphorus in the diet exercise an unfavorable influence on the calcium metabolism, and are accompanied by an increase in the calcium lost in the feces.

3. The retention of one element in the intestine by an excessive amount of the other in the diet is best explained by the formation of insoluble phosphates of calcium which cannot be absorbed.

THE PERISTOLIC FUNCTION

ITS PRACTICAL SIGNIFICANCE IN CASES OF PYLOROSPASM AND HABITUAL VOMITING *

JULIAN L. ROGATZ, M.D.
NEW YORK

Recent efforts to cure cases of pylorospasm and habitual vomiting have been based on the use of thick cereals, or otherwise concentrated feedings of thick consistency. Though empirical, this method of feeding has given so many satisfactory results that one is now always willing to try a conservative treatment with cereals before resorting to a radical procedure such as an operation. The material offered in this paper will suggest a scientific explanation for the success of this important therapy.

REVIEW OF THE LITERATURE

This modern trend in the treatment of cases of infantile vomiting is evident in the literature of recent years. Sauer,[1] in discussing pyloric stenosis, mentions the very unpleasant and unavoidable complications due to the formation of bands of adhesions, which result from operation. In contrast, he quotes the excellent results obtained with thick cereal feedings: "In favorable cases the vomiting stops and the gain in weight is gratifying." Sauer's report contains thirty-five cases of pyloric stenosis, of which twenty-eight patients were treated successfully with thick cereal. Of the remaining seven operated on, five recovered and two died.

Lust [2] says that in severe cases of habitual vomiting one may have prompt success with any kind of thick gruel. Huldschinsky [3] reported a case of rumination, which was treated with equal success by using a thick gruel prepared with half milk, after all other measures had failed. Mayerhofer [4] discusses a similar case in a child with inanition, aged 7 months, wherein the persistent rumination and vomiting were controlled most satisfactorily and promptly by feeding a thick cereal.

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* Received for publication, Sept. 15, 1924.

* The last of a series of three papers, devoted to the roentgenographic study of stomach function in infants.

1. Sauer, L. W.: A Further Contribution to the Study of Pyloric Stenosis, Am. J. Dis. Child. **22**:166 (Aug.) 1921.

2. Lust, F.: Zur Pathogens der Rumination im Sauglingsalter, Monatschr. f. Kinderh. **10**:316, 1912.

3. Huldschinsky, K.: Zur Therapie der Rumination in Sauglingsalter, Ztschr. f. Kinderh. **8**:363, 1913.

4. Mayerhofer, E.: Zur Therapie der Rumination in Sauglingsalter, Therap. Monatsh. **26**:263, 1912.

In this connection I wish to summarize the record of a striking case, which appeared at the Kaiser and Kaiserin-Friedrich Krankenhaus in Berlin during the work on peristolic function [5] and quite independently thereof. The child, aged 6 months, was admitted to the infant service on August 24 with a diagnosis of dysentery. The patient weighed 4,700 gm. (10.3 pounds). There was no fever. The stools were loose and slimy. He ruminated a great deal and began to lose weight. There was a slight rise in the temperature (Fig. 1). As the temperature became normal the weight increased, but finally pyuria developed and the child lost weight again, so that on September 27, more than one month after admission, the patient weighed 200 gm. less than when admitted. At this point it was decided to combat the constant regurgitation, which persisted throughout, by using a moderately thick, half milk gruel before each feeding. At once the rumination stopped. The

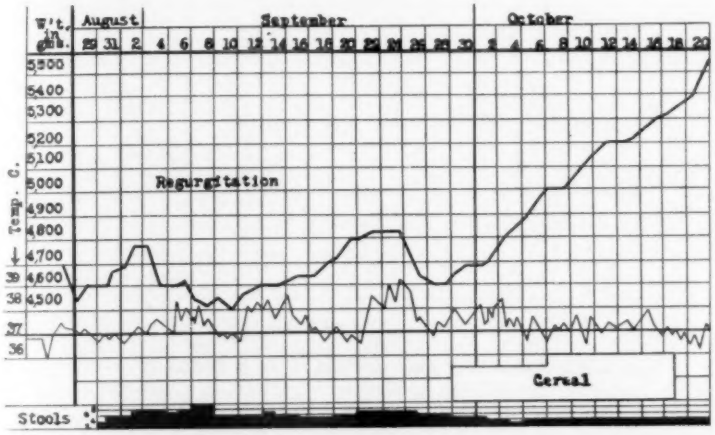

Fig. 1.—A case of persistent regurgitation, showing marked improvement in weight and stools following the use of thick cereal (two teaspoonfuls before each feeding). The formula used up to September 28 was concentrated albumin milk.

stools were almost normal. The child began to gain and continued thus until discharge, one month later, when he weighed 5,500 gm. (12.10 pounds). The chart of this patient illustrates the striking efficacy of thick gruels.

Finkelstein had an even more striking case in a child, aged 8 months, who vomited persistently for a period of several months. Various food combinations with moderately thick gruels resulted in short periods of improvement with consequent gain in weight (Fig. 2). Still the

5. Rogatz, J.: Roentgen-ray Studies of Stomach Function, Am. J. Dis. Child. **28**:52 and 69 (July) 1924.

vomiting never ceased entirely. There was occasional relief for one or two days, but then the symptoms began anew, and with each spell of increased vomiting there appeared a rapid decline in weight. At one time mashed potato and concentrated whole milk gruel were combined in equal parts, giving a feeding of thicker consistency than those hitherto used. Despite a more marked improvement than ever before, vomiting became more frequent again and a gradual but steady loss of weight set in. Finally, on the basis of the demonstration that gruels prepared with milk lose their consistency in the stomach and thus prevent the most efficient action of the peristolic function,[5] it was decided to prepare a feeding as thick in consistency as possible. This was accomplished by increasing the mashed potato in quantity. The milk diluent was decreased to its lowest point but made more concentrated by the addition

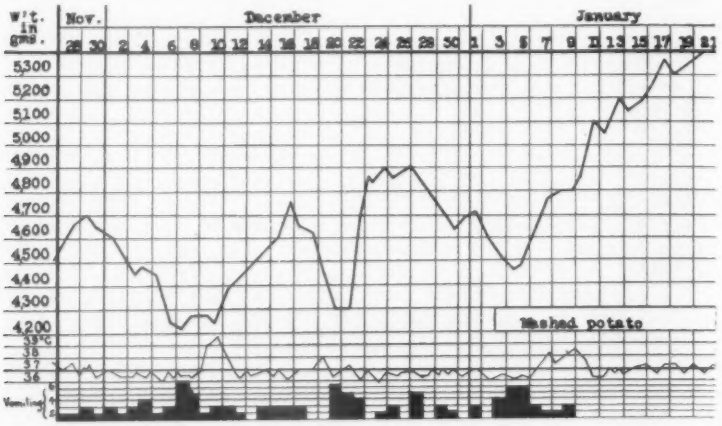

Fig. 2.—A case of continuous vomiting cured with thick, mashed potato. The formulas used were as follows: November 27, whole milk gruel, seven feedings and vegetable puree one feeding; December 7, eight feedings of concentrated albumin milk gruel with 20 per cent. sugar, 1½ ounces each feeding; December 20, 10 ounces mashed potato diluted with 10 ounces milk gruel; January 3, 16 ounces mashed potato, 6½ ounces milk gruel with 10 per cent. protein milk powder. With this increase in mashed potato the vomiting ceased and the weight increased without further trouble.

of protein. From this time recovery was complete. The vomiting stopped and the gain in weight was rapid and steady. This improvement continued for three weeks, after which, there having been no relapse, the child was discharged perfectly well. It is very likely that if the food had been thickened earlier and if less of the milk diluent had been used, the cure would have been even more striking. However, it was deemed wiser to be cautious. The chart is most instructive and is here presented.

Finally, Ylppö [6] described the interesting and significant case of a child, who vomited constantly on a normal fluid diet, and lost weight as a result, until thick cereals of half milk with 5 per cent. of wheat flour and cane sugar were given. At once the vomiting ceased and a gain in weight began. On several occasions a return to fluid diet was accompanied by renewed vomiting and loss of weight. The progress of this case under the respective influences of fluid and cereal diets is well illustrated in the patient's fluctuating weight curve, which appears in Ylppö's paper.

Mixsell [7] has reported his success with thick cereals in cases of malnutrition in infants, and he explains the unexpected tolerance of the child for the high carbohydrate diet in such cases on a chemical basis. The different sugars used are absorbed one at a time, with varying degrees of rapidity, thus enabling the weakened organism to utilize its carbohydrate without being upset by an overdose.

He further states: "The more complicated the structure of a carbohydrate, the more numerous the steps in its breaking down to the end-product. There is thus less fermentable material in the intestine at one time and less opportunity is afforded for fermentation to get the upper hand."

One problem must still be solved. Why do thick cereals pass into the duodenum in cases of pylorospasm and habitual vomiting while the same food, thinned to reduce consistency, is promptly vomited? In the recent roentgenologic work on stomach function, described in two earlier papers,[5] it was determined that the infant stomach contracted down strikingly around a content of thick consistency. This actual demonstration of what is termed "peristolic function" has an important, practical significance. By virtue of this function, the evident superiority of thick cereal feedings in appropriate cases may be explained on a more than purely empirical basis. Toward this end it will be necessary to discuss the mechanism of vomiting in some detail.

Vomiting in the infant is very often mechanical in nature. Sometimes it is caused by the quality of the feeding, too much sugar, or excess fat, etc.; other times by some toxic irritation of the central nervous system or of the fibers of the muscle wall directly. Whatever the impulse, however, the actual act of vomiting only takes place as a result of a muscle contraction of the stomach wall.

The mechanical explanation of vomiting as a basis for therapy becomes particularly significant in cases of habitual vomiting. Among

6. Ylppö, A.: Ueber Rumination mit Luftschlucker beim Sauglingen, Ztschr. f. Kinderh. **15**:84, 1916.

7. Mixsell, H. R.: Further Studies in Thick Cereal Feeding in Malnutrition in Infancy, Arch. Pediat. **37**:490 (Aug.) 1920.

the causes of habitual vomiting, Finkelstein [8] notes (1) air swallowing, in which condition the child swallows much air, causing a marked distention of the stomach, with consequent belching, during which the infant regularly brings up part of its food; (2) an atonic state of the entire body musculature, part of which atony manifests itself in the stomach. A slight pressure on the abdomen, such as might be caused by lifting the child or otherwise changing its position, may suffice to cause a cardiospasm and consequent vomiting; (3) a hypertonic group, classified as having a constitutional hypersensitiveness of the stomach, really a hyperesthesia of the mucous membrane, and an increased irritability of the muscular wall (Lust [9]), common in neuropathic and hypertonic infants, and (4) a group of children, who ruminate or regurgitate, which Finkelstein considers similar to cases of "nervous" cardiospasm in symptomatology.

Thus, the vomiting in pylorospasm and in the various types of habitual vomiting described may be explained on a mechanical basis of muscular wall contraction. It should be possible to control the vomiting in the foregoing types of cases by stimulating the peristolic function of the stomach. This, as has been shown experimentally, can be done by feeding thick cereals or other appropriate foods of thick consistency.

COMMENT

An explanation of the mechanical principles involved in stomach motility will tend to justify these conclusions. In an article on gastric motility, Kreuzfuchs [10] explained that the stomach divides itself functionally into two parts, the larger, upper part (the corpus ventriculi) and the lower, pyloric antrum. The upper portion breaks up the food and the lower segment prepares it for further digestion and emptying into the duodenum. In this activity the fundus changes its form, while the pyloric region retains its shape. Kreuzfuchs further pointed out that it is not always a peristaltic motion which propels the food, but a "different, peculiar, pressing together of the walls, a sort of closing in on the food, which often results in the contents being directed toward the fundus rather than toward the pylorus."

This suggests the peristolic function. Just as soon as food enters the stomach the muscular walls are stimulated to contraction. Occuring simultaneously with the familiar, peristaltic action the impulse appears to surround and grasp the food content (peristolsis). In a fluid medium

8. Finkelstein, H.: Lehrbuch der Sauglingskrankheiten, Ed. 2, Berlin, Julius Springer, 1921, p. 669.

9. Lust, F.: Diagnostik und Therapie der Kinderkrankheiten, Ed. 3, Berlin, Urban and Schwarzenberg, 1923, p. 118.

10. Kreuzfuchs, S.: Die Magenmotilitat in radiologischer Beleuchtung, Wien. med. Wchnschr. **62**:1070, 1912.

this is difficult because of the inherent, elusive nature of fluids, which tend to travel away from the source of pressure. Hence, this peristolic effort results in vomiting of the gastric content in some children. If a thick cereal feeding is used in these cases, the stomach is enabled to exert its peristolic effect more successfully. Since less air is swallowed, the stomach content is correspondingly decreased and the organ has a greater surface area to devote to surrounding and grasping its food.

In patients where a great deal of air is swallowed with cereal, as occasionally happens, the muscular action of the stomach merely results in expulsion of the air. At the same time the stomach wall surrounds and retains the thick food, which is less mobile than fluid and offers a welcome resistance to the gastric musculature. This is proved by the fact that the organ with a thick cereal almost always assumes an oval or circular form, regardless of size. We have never seen a large fundus and a long thin pylorus (Alwens and Husler [11]) with cereals, but always, as our plates indicate,[5] the small, circular organ, with very little or commonly no air bubble at all.

It is very probable that future investigation will disclose the variable tendencies of different gastric organs to react to foods of varying consistency. It is likely that some stomachs will exert a more powerful and successful peristolic effort with a food of given thickness than other stomachs. It follows that some infants may require a thicker food than others in order to elicit peristolsis and thus stop vomiting.

Thus, in conclusion, one may explain the favorable result with cereals, in cases of vomiting, on the basis of this mechanical relation, which exists between the stomach contents and the muscular wall of that organ. It matters not whether the child is neuropathic or whether there is a pyloric spasm, this relation must obtain in every stomach, and to win a proper balance of forces, such as is possible with thick cereal feedings, is the logical means of successful treatment.

SUMMARY

1. Recent success in the treatment of cases of habitual vomiting and pylorospasm with thick cereals justifies the effort made in this paper to establish this method of therapy on more than merely empirical grounds.

2. Two typical cases and their charts are presented, which indicate the striking results obtainable with thick cereal feedings.

3. A mechanical explanation for vomiting in pylorospasm, and in habitual vomiting due to various causes, is offered as the logical basis for a thick cereal therapy in these cases.

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11. Alwens-Husler.: Roentgenuntersuchungen des kindlichen Magens, Fortschr. a. d. Geb. d. Röntgenstrahlen. **19**:3, 1912.

4. This mechanical basis for vomiting in infants is directly associated with the existence of a peristolic function, which manifests itself in the infant stomach as a capacity for surrounding and grasping a food content of thick consistency.

5. The practical significance of the peristolic function in the infant stomach is discussed, so far as it is directly responsible for the successful use of thick cereals.

CONCLUSIONS

1. A definite, mechanical relation exists in the infant stomach between its food content and the stomach wall. By direct contact, the food ingested stimulates the muscle wall to surround and grasp its content (termed peristolic function).

2. The vomiting in pylorospasm and in various types of habitual vomiting may be explained mechanically, on the basis of muscular wall contraction.

3. It should be possible to control vomiting in the before mentioned types by stimulating the peristolic function of the stomach.

4. This can be accomplished practically by the use of a thick cereal or similar food of appropriate thickness.

ACUTE HEMORRHAGIC NEPHRITIS AND ACUTE HEMORRHAGIC ENCEPHALITIS FOLLOWING VARICELLA

REPORT OF A CASE WITH RECOVERY *

MURRAY B. GORDON, M.D.
BROOKLYN

Varicella is generally characterized by a mild clinical course without any complications. This case is reported because it presents unusually bizarre and severe complications.

REPORT OF CASE

V. H., a boy, aged 6 years, was admitted to the United Israel Zion Hospital on May 3, 1923, referred by Dr. John Hirsch. There was a previous personal history of convulsions at 2 years of age with "teething," pertussis at 5 years, measles at 5½ years and tonsillectomy at 4 years. The family history was negative.

According to Dr. Hirsch, the boy developed varicella two weeks prior to admission. The clinical course was very mild and the lesions were typical, not very numerous but generally distributed over the scalp, face and body. Three days prior to admission, there was a slight rise in temperature and headache and polyuria with hematuria the next day.

Physical Examination.—On admission, a poorly nourished boy of the tall, thin type who did not appear acutely ill or distressed, was seen. He was alert, bright and spoke distinctly in a clear voice. Numerous typical lesions of the last stages of varicella were present on the scalp, face and body. No further abnormal findings were elicited on routine physical examination. The blood pressure was 90 systolic, 62 diastolic; the temperature was 98, respirations 22, pulse 120. Urinalysis revealed a urine that was turbid, acid and reddish in color, with a specific gravity of 1,026, with a large amount of albumin, numerous red and white blood cells, moderate number of granular and hyaline casts and no sugar or acetone. Diagnosis: Acute hemorrhagic nephritis following varicella.

The boy was placed in the isolation room. For the next two weeks there was an increasing edema, appearing first on the face and then involving the ankles, hands and scrotum. There was frequent vomiting. The blood pressure averaged 100 systolic and 76 diastolic. The blood findings showed carbon dioxid combining power of 65 c.c. to 100 of blood. Urea nitrogen, 41.6 mg. per hundred cubic centimeters; creatinin, 2.7 mg. per hundred cubic centimeters; uric acid, 4 mg. per hundred cubic centimeters; glucose, 1.25 per cent. The cell count showed 4,000,000 red blood cells, 8,600 leukocytes, 68 per cent., polymorphonuclears, 12 per cent., large lymphocytes and 20 per cent. small lymphocytes. The Wassermann and Pirquet tests were negative.

On May 23, the boy complained of dizziness and headache. At about 10:30 a. m. he was seized suddenly with a convulsive attack which was followed in rapid succession by several similar ones, eclamptic in type. He remained unconscious all day, his face cyanotic, pupils dilated, pulse weak. His face and extremities were in a state of almost constant spasmodic twitchings. The temperature jumped from 99 to 106 with a corresponding rise in pulse, respira-

* Received for publication, Aug. 15, 1924.
* From the Pediatric Service of the United Israel Zion Hospital.

tion and blood pressure. He responded to treatment, which consisted of venesection, lumbar puncture, colonic irrigation, rectal drip of glucose and bicarbonate of soda, hot packs and stimulation. The next morning a right facial paralysis was noted, and as he became more conscious it was apparent that there was also a loss of speech and hearing and loss of motion in the right arm and leg.

A neurologic examination by Dr. Simon Rothenberg on May 26 revealed that the boy was still in a semiconscious state. Motor aphasia and apparent deafness were present. The pupils reacted to light; fundus examination showed engorgement of the blood vessels but no degenerative changes. There was an involvement of the seventh cranial nerve on the right side, no involvement of the fifth on either side and no eye muscle involvement. The right upper and lower extremities showed a hemiplegic condition with loss of reflexes and some loss of pain sensation on that side. Babinski and Oppenheim reflexes were present on the right. Diagnosis: Hemiplegia of right side due to a lesion of the cortex of the left side of the brain, hemorrhagic lesion in Broca's area.

Complete consciousness was present for the first time on May 30. The abdomen showed a fluid wave, and edema still persisted in the hands and feet. The blood pressure ranged between 112 and 116, reaching 120, however, at one time. There was little change in the blood constituents, the carbon dioxid combining power being 58 c.c. and urea nitrogen 62.5 mg. There was a rise in the creatinin to 5 mg. per hundred cubic centimeters. The phenolsulphonphthalein test, which could not be performed previous to this because of the bloody urine, revealed on June 1, 15 per cent. in the first hour and 10 per cent. for the second hour, a total of 25 per cent. A fundus examination by Dr. I. Kruskal gave negative findings. Because of the low hemoglobin, a transfusion of 100 c.c. of his brother's blood (Group 2) was performed.

There was a gradual improvement, the urinary output increasing from 15 to 60 and 70 ounces a day. The urinary findings averaged a specific gravity of 1,006, a color which varied from a smoky red at the onset to a clear amber later, a marked increase in albumin, many granular and hyaline casts and numerous red and white blood cells. The treatment had consisted of two hot packs a day, restriction of fluids, colonic irrigations of bicarbonate of soda, imperial drink, magnesium sulphate in dram doses every hour for four doses each morning until improvement was noted, and diuretin and digitalis occasionally.

The boy was discharged from the hospital on June 23. He had regained the use of his legs and was able to walk a short distance with effort, dragging the right leg. There was a general weakness of the right upper extremity evidenced by a drooping of the shoulder, difficulty in raising the arm and weakness of the hand. The face still showed a right sided paralysis; both pupils were dilated, reacting to light and in accommodation. He heard when spoken to, but would answer as if with an effort in a very slow, almost indistinct, low pitched monotone. Correlation was still sluggish. The elbow reflexes and knee jerk were sluggish on the right but on that side all other reflexes were normal.

Laboratory Findings.—Phenolsulphonphthalein test; 15 per cent. for the first hour, 16 for the second, a total of 31 per cent. Urinalysis: Specific gravity, 1,010, acid in reaction, yellow in color, traces of albumin, no casts, no sugar, several red and white blood cells. Blood pressure: systolic, 90, diastolic, 70. Blood constituents: urea nitrogen, 31 mg. per hundred cubic centimeters, creatinin, 1.37 mg. per hundred cubic centimeters.

Course after Discharge from the Hospital.—The physical disorders gradually improved so that by October 20, there was an almost complete recovery of the facial paralysis and of the hemiplegia. The grip in the right hand was still weak and he was unable to grasp a pencil with all his fingers, holding it with his thumb and forefinger. The mental condition did not show the same degree of improvement, as there was still a sluggishness in correlation and difficulty in the formation of words and sentences. In addition to this there

was an impairment of memory and a change in disposition and behavior, presenting a typical picture of behavior and conduct disorder. Instead of the almost model child prior to the onset of varicella, he was now mischievous, disobedient, untruthful, unmanageable and subject to tantrums and outbursts of bad temper. The renal condition had cleared, showing normal urine and blood findings.

COMMENT

While we cannot definitely eliminate all possible factors capable of producing acute hemorrhagic nephritis at any time, we are justified in the present case to consider that the attack of varicella was the sole etiologic factor, especially since there was no history of acute tonsillitis, sore throat or any other acute illness prior to the attack of chickenpox.

Wilcox and Lyttle [1] have recently demonstrated that there is an impairment of kidney function with nitrogen retention in most cases of acute infectious processes. That nephritis can complicate varicella was first pointed out by Henoch. Its occurrence, however, is so infrequent that Ker [2] of Edinburgh states that he has seen but two cases of true nephritis complicating chickenpox. I have not seen a single example of this complication in over ten years connection with the Kingston Avenue Hospital for Contagious Diseases. When present, according to Cerf and Unger,[3] the nephritis may take one of three forms: (*a*) latent nephritis in which there are no symptoms and albuminuria is discovered only on investigation; (*b*) mild nephritis in which there is marked albuminuria and some edema but no severe symptoms, and (*c*) severe nephritis with fever, marked albuminuria, vomiting, gastrointestinal disturbance, uremia, etc.

The inter-relationship between nitrogen retention, high blood pressure, coma and convulsions has been the subject of numerous controversies which cannot be entered into here. But it is admitted that convulsions, coma, amaurosis, transient paralyses and delirium are due to causes other than nitrogen retention alone. Vollhard [4] and the German school uphold Traube's theory advanced many years ago that these are end-results which are produced by cerebral edema. Mosenthal,[5] on the other hand, denies that cerebral edema has any bearing on these conditions, claiming that they are due to pathologic changes in the cerebral arteries occurring as an affection entirely independent of the

1. Wilcox, H. B., and Lyttle, J. D.: Kidney Function in Acute Disease, Am. J. Dis. Child. **26**:195 (Sept.) 1923.

2. Ker, C. B.: Infectious Diseases, Ed. 2, London, Oxford University Press, 1920, p. 220.

3. Quoted by Swoboda, in Pfaundler, M., and Schlossmann, A.: The Diseases of Children, Philadelphia, J. B. Lippincott Company, **2**:330, 1912.

4. Vollhard and Fahr: Die Brightsche Nierenkrankheit, 1914.

5. Mosenthal, H. O., in Barker, L. F.; Hoskins, R. G., and Mosenthal, H. O.: Endocrinology and Metabolism, New York, D. Appleton & Co., Chapter on Metabolism in Nephritis, **4**:382-396, 1923.

renal condition. He is upheld in his contention by Feinblatt,[6] who concluded, as a result of a study of uremia, that convulsions should be considered not as a part of the picture of chemical uremia but as an indication of some pathologic cerebral condition. The latter further states that an indefinite hemiplegia in a comatose patient with nephritis has often been classed as an evidence of uremia until the necropsy disclosed cerebral hemorrhage.

The symptom complex presented by this case is open to interpretation from these two different angles.

The first, in keeping with the views of Traube, would be based on the assumption that the entire clinical picture was the end-result of a nephritic toxicosis going through the various and successive stages of acute hemorrhagic nephritis, nitrogen retention, chemical uremia, coma, convulsions, hemiplegia and finally mental and psychic changes due to organic brain lesions following on hemorrhagic softening.

The second point of view would be predicated on the supposition that the clinical syndrome was due to two distinct and different pathologic lesions, one renal and the other cerebral, both produced, however, by the same underlying etiologic factor, the original disease, varicella. While the nephritic toxicosis would be responsible for the nephritis, nitrogen retention and possibly also the coma, the cerebral disturbance would be responsible for the convulsions, hemiplegia and the subsequent conduct disorder.

That this latter view is in all probability the correct one is strengthened by a comparison of the symptomatology of this case with one recently reported by Toomey, Dembo and McConnell.[7]

Their patient was a boy, aged 6 years, who developed a similar condition twenty days subsequent to an attack of scarlet fever, the symptoms consisting of fever, vomiting, squint, coma, Cheyne-Stokes respiration, cyanosis and death. He was admitted to the hospital on a diagnosis of post-scarlatinal nephritis and uremia and died forty-five minutes after admission. At necropsy, a congestion of the right kidney was found on inspection; no microscopic examination was made. The brain on gross inspection showed a hemorrhagic encephalitis with intraventricular hemorrhages; microscopically it revealed thrombosis of the cerebral vessels accompanied by edema and hemorrhagic infiltration.

Hoppe [8] states that encephalitis of this type may occur after measles, scarlet fever and diphtheria, Putnam [9] reported one after mumps. The

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6. Feinblatt, H. M.: Uremia: The Syndrome of Nitrogen Retention, Boston M. & S. J. **189**:399-405 (Sept. 20) 1923.

7. Toomey, J. A.; Dembo, L. H., and McConnell, G.: Acute Hemorrhagic Encephalitis: Report of a Case Following Scarlet Fever, Am. J. Dis. Child. **25**:98-107 (Feb.) 1923.

8. Hoppe, in Tice: Practice of Medicine, Hagerstown, Md., W. F. Prior & Co. **9**, 1920.

9. Putnam, J. J.: J. Nerv. & Mental Dis. **24**:1897.

encephalitis may be produced either by the direct bacterial action of the bacteria responsible for the original disease or by the toxemia of the acute infection.

Patients who recover are left with cortical defects resulting from the fibrotic changes and the formation of scar tissue. According to Leahy and Sands,[10] the replacement of the destroyed nerve tissue by neuroglia scar tissue acts both as a stimulating and irritating agent to the rest of the nerve tissue. They explain the motor and sensory disturbances and conduct disorders in encephalitis to such cerebral changes. They also found that patients who had made an apparently complete somatic recovery not infrequently manifested some striking forms of abnormal behavior. In the present case there was a post-encephalitic syndrome of impaired memory, sluggish correlation, altered disposition and impaired speech. Their observation that recovery takes place in the somatic elements and then in the psychic is borne out in this case.

SUMMARY AND CONCLUSIONS

A boy, aged 6 years, following a mild attack of varicella, developed hematuria, nitrogen retention, coma, convulsions, right hemiplegia, right facial paralysis, loss of speech and of hearing. He recovered his speech and hearing and made an almost complete recovery from the paralysis but later showed mental and psychic changes.

It is felt that there were two distinct and different pathologic conditions present, acute hemorrhagic nephritis and acute hemorrhagic encephalitis, the major part of the cerebral hemorrhage occurring in Broca's area.

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10. Leahy, Sylvester R., and Sands, Irving J.: Mental Disorders in Children Following Epidemic Encephalitis, J. A. M. A. **76**:373-377 (Feb. 5) 1921.

HEART BLOCK IN A CHILD OF TEN YEARS FOLLOWING TRAUMA TO THE PRECORDIUM *

WILLIAM ROSENSON, M.D.
NEW YORK

During the last ten years, the more general use of the electrocardiograph and the intensive study of cardiac conditions in children have revealed disturbances of rhythm and conduction in a considerable number of patients.

Heart block is one of the less common forms of abnormal cardiac mechanisms found in childhood. In 1922 I was able to collect thirty-six cases of heart block in infancy and childhood from the literature, and added one observed in a girl, aged 11 years, with a congenital cardiac lesion.[1] The etiologic factors in the cases described were as follows: Diphtheria, sixteen cases; rheumatism, two cases; congenital syphilis, one case; cardiac tumor, one case; nose and throat infection, one case; associated congenital cardiac lesion, twelve cases and unknown cause, three cases.

Heart block occurring in patients during the course of diphtheria present the gravest outlook, the mortality in the collected cases having been 93.7 per cent.

The following case of heart block is unique, because of the very unusual etiology and the rapid recovery.

REPORT OF CASE

Present History.—I. B., a boy, aged 10½ years, three years before this examination, while playing outdoors received a hard fist punch over the precordium, inflicted by one of his playmates. Following the blow, he complained of weakness and a peculiar fluttering sensation over the region of the heart. He was unable to walk, felt faint and was carried to bed. Two days later, on attempting to walk, he fell and was unconscious for a few minutes. There was slight dyspnea on exertion, no vomiting or other gastro-intestinal disturbances. No fever. No antecedent infection.

Family History.—Negative. There were no other children nor was there any history of miscarriages.

Past History.—The child was born at full term, of instrumental delivery. Birth weight was 7 pounds (3.2 kg.). The infant was breast fed. Diseases of infancy included scarlet fever at the age of 3 years, measles at 5 years and whooping cough at 6 years. There was no history of diphtheria or rheumatic infection.

Physical Examination.—The child appeared well nourished but pale. There was slight dyspnea. Examination of the heart showed no enlargement. There

* Received for publication, Oct. 10, 1924.

1. Arch. Ped. Vol. XXXIX No. 9, Sept., 1922.

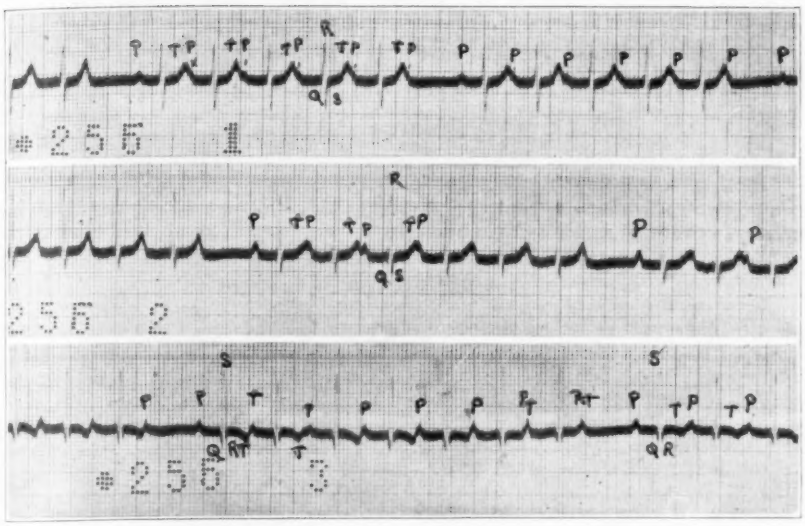

Fig. 1.—Various grades of heart block following trauma to the chest wall.

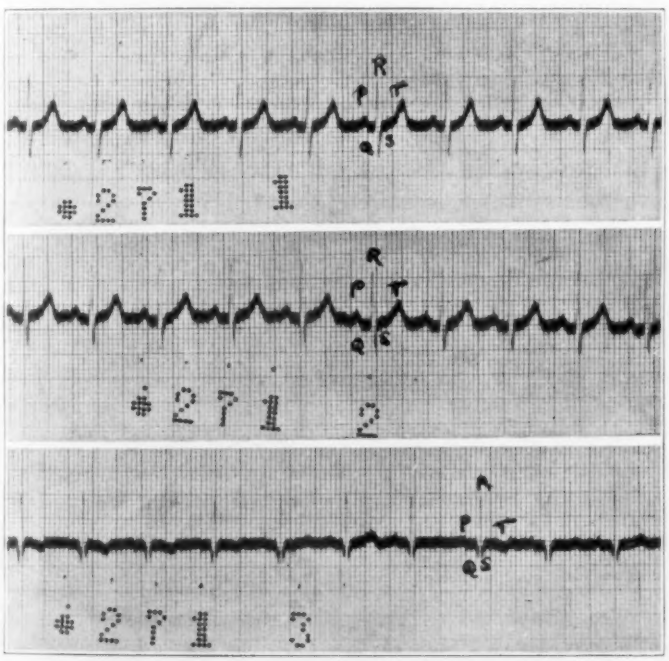

Fig. 2.—Normal rhythm electrocardiogram taken three months later.

was a systolic murmur, best heard over the pulmonic area, functional in character. The heart sounds were of normal quality. The pulses were equal, rapid and irregular, of fair volume. There was no pulse deficit. The rate was 120. The rest of the physical examination was negative.

Laboratory Findings.—Urine was normal.

Blood Counts.—Hemoglobin, 75 per cent.; red blood cells, 4,550,000; white blood cells, 8,400; polymorphonuclears, 70 per cent.; small lymphocytes, 25 per cent.; large lymphocytes, 5 per cent. Blood pressure was systolic 80, diastolic 45. The Wassermann reaction was negative.

The electrocardiographic study (Fig. 1) was made by Dr. Marcus Rothschild.

The electrocardiogram showed varying grades of heart block, with a rapid ventricular rate.

The conduction time between auricle and ventricle, as indicated by the P-R interval, was prolonged, measuring 0.28 of a second.

The P and T waves usually fall together; in certain parts of the tracing they are distinctly separate, and in the tracing marked 256, Lead III, they can easily be distinguished, as the T wave is inverted and the P wave upright. Where they happen to fall together an upright wave results, as the voltage of the P is greater than the voltage of the T.

Throughout the tracing there are many periods where one auricular beat is completely blocked, giving periods of two to one block, varying with periods of prolonged conduction.

Lead I is diphasic, which possibly points to some predominance of the right ventricle.

Treatment.—The child was kept in bed for about two weeks and made a complete recovery. No specific medication was employed. The electrocardiogram returned to a normal rhythm (Fig. 2).

During the last three years the child has had no recurrence of the condition, and his physical health has been normal.

COMMENT

This case is unusual because of the etiologic factor. The pathologic process that developed in the conduction system following the blow to the chest wall is obscure. It may have been a slight hemorrhage in or about the node of Tawara or a functional change in the nervous mechanism. There has been no similar case recorded in infancy or childhood.

PROGRESS IN PEDIATRICS

A REVIEW OF THE LITERATURE ON WHOOPING COUGH FOR THE LAST FIVE YEARS, FROM 1919 TO 1924 *

LAWRENCE W. SMITH, M.D.

BOSTON

Something over 300 papers have appeared in the last five years on whooping cough, most of which are relatively unimportant from the point of view of any increase in knowledge, concerning either the etiology or the diagnosis of the disease. There have been several articles dealing particularly with the treatment by means of intramuscular injection of ether, by vaccines and by the roentgen ray, which seem of some value.

WHOOPING COUGH IN NEW-BORN INFANTS

Phillips [1] reports two patients infected by the obstetrical nurse, who was in the catarrhal stage of the disease at the time of delivery. Both of these cases occurred in female infants, who began coughing on the eighth and eleventh day. One of these babies died, the other lived after a stormy convalescence.

WHOOPING COUGH IN INFANTS

Gottlieb and Möller [2] give an elaborate differential discussion of whooping cough in infants as compared to older children. In an epidemic occurring during 1922, they found that the incubation period was usually at least three weeks, and sometimes as long as five and one-half weeks in several instances. The degree of infection is considerably greater than in other upper respiratory infections, including tuberculosis, and isolation is strongly urged. They report a case of whooping cough occurring in a young breast-fed baby, which contradicts the theory of Neumann, that babies nursed by mothers who have had whooping cough are immune.

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* Received for publication, Aug. 13, 1924.

* From the Boston Floating Hospital.

1. Phillips, John: Whooping Cough Contracted at Time of Birth with Report of Two Cases, Am. J. M. Sc. **161**:163 (Feb.) 1921.

2. Gottlieb, K., and Möller, B.: Whooping Cough in Infants, Jahrb. f. Kinderh. **100**:222-236 (Dec.) 1922.

WHOOPING COUGH IN ADULTS

Schwenkenbecher,[3] Neurath [4] and Hennes [5] report cases of whooping cough occurring in adults, and suggest that the disease is much more frequent than commonly supposed, as there is unconscious inhibition of the spasmodic cough, which obscures the diagnosis.

GENERAL

Talbot [6] summarizes the present status of whooping cough in a clinic given at the Massachusetts General Hospital. Herrman and Bell [7] report a careful study of 300 cases of pertussis with reference to age, sex, seasonal incidence, the relation to other infections, particularly tuberculosis, and treatment in a hospital as compared to home care. They recommend immunization of infants by pertussis vaccine in order to secure control of the disease, and a reduction in its mortality. This is one of the best summaries of the subject encountered in the literature.

ETIOLOGY AND BACTERICLOGY

There are a number of articles dealing with the etiology of the disease. Cozzolino [8] denounces Czerny's theory of a neuropathic predisposition as being the etiologic factor in producing the whoop, and in an outbreak which he studied came to the conclusion that it was due to the specific infection by the *Bacillus pertussis*.

Meyer [9] in several articles reports the value of the bacteriologic diagnosis of whooping cough. At the Serum Institute at Copenhagen he has obtained in 1,106 cases of whooping cough, 970 positive cultures of the *Bacillus pertussis*. In 120 cases during the catarrhal stage he obtained 75 per cent. positives. In the first week of the paroxysmal cough he obtained 57 per cent. positives in 277 cases, and in the second

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3. Schwenkenbecher, A.: Whooping Cough in Adults, Med. Klin. **17**:1447 (Nov. 27) 1921.

4. Neurath, R.: Whooping Cough in Adults, Med. Klin. **17**:1173 (Sept. 25) 1921.

5. Hennes, H.: Whooping Cough in Adults, Med. Klinik **17**:591 (May 15) 1921.

6. Talbot, F. B.: Whooping Cough, M. Clin. N. America **3**:1127 (Jan.) 1920.

7. Herrman, C., and Bell, T.: Study of Three Hundred Cases of Pertussis in a Hospital, Arch. Pediat. **41**:13-28 (Jan.) 1924.

8. Cozzolino, O.: Whooping Cough, Pediatria **28**:113 (Feb.) 1920.

9. Meyer, A. H.: Researches on Whooping Cough in the State Serum Institute, Ugeskr. f. Laeger **83**:523 (April 21) 1921; Laboratory Diagnosis of Whooping Cough at Serum Institute of State, Acta. Paediat. **1**:99 (March) 1921; Laboratory Tests for Pertussis, Acta. Paediat. **1**:99, 1921; Bacteriology of Whooping Cough, Ugesk. f. Laeger **83**:523 (April 21) 1921; Report of Mme. Chievitz and Dr. A. H. Meyer, Copenhagen, Ann. de L'Inst. Pasteur **30**:503, 1916.

week of the cough 61 per cent. positives in 201 cases. After the fourth week he obtained 9 per cent. positives in 107 cases. He further recommends the examination of the blood serum for complement fixation, which occurs regularly about the third week of the disease. This diagnostic service is conducted by sending out, on request, special aluminum containers with suitable culture mediums to physicians. These are inoculated by coughing directly into them at a distance of 6 inches, and are then returned to the Institute by mail.

Povitzky [10] reports an improvement in the technic for the isolation of the *Bacillus pertussis* by acidifying the medium to a hydrogen-ion concentration equivalent to p_H 5. By this method she has secured positive results in 38 per cent. of twenty-six cases as compared to 22.8 per cent. in her first series of thirty-five cases.

Krumwiede, Mishulow and Oldenbusch [11] find that by agglutination and agglutin absorption methods two serologic groups, A and B of *Bacillus pertussis,* exist. Most of the cases in the epidemic they studied belonged to group B.

Barbier and Renard,[12] and Lereboullet [13] report that cases of whooping cough are rarely positive culturally after four weeks, and that the contagious stage of the disease is practically restricted to the catarrhal stage.

STATISTICS

Young [14] has made an exhaustive study concerning the periodicity of pertussis epidemics from 1870 to 1910 in England and Scotland, and comes to the conclusion that the periodicity has too definite a cyclic appearance to be due to accident. The periodicity is due to the season, to differences in the organism and to the interrelationship of coincidental epidemics of measles and pertussis.

Henry,[15] in reporting a statistical study of measles and whooping cough in Massachusetts from 1913 to 1919, brings out the point that

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10. Povitzky, O. R.: Improved Methods for the Isolation and Cultivation of B. pertussis, J. Infect. Dis. **32**:8, 1923.

11. Krumwiede, C.; Mishulow, L., and Oldenbusch, C.: The Existence of More Than One Type (Immunologic) of B. pertussis, J. Infect. Dis. **32**:22, 1923.

12. Barbier, H., and Renard: Contagiousness of Whooping Cough, Bull. et mém. Soc. méd. d. hôp. de Paris **46**:1592 (Dec. 1) 1922.

13. Lereboullet, P.: Contagious Phase of Whooping Cough, Paris méd. **9**: 44 (Jan. 11) 1919.

14. Young, M.: Investigation Into Periodicity of Epidemics of Whooping Cough from 1870 to 1910 by Means of Periodogram, Proc. Roy. Soc. Med. (Sect. Epidemiology) **13**:207 (May) 1920.

15. Henry, J. E.: Statistical Study of Recent Experience with Measles and Whooping Cough in Massachusetts, Am. J. Pub. Health **11**:302 (April) 1921.

33 per cent. of the cases and 90 per cent. of the deaths of whooping cough occur in children under 3 years, and recommends that public health propaganda be directed toward vaccine prophylaxis, aimed particularly at this preschool age. In this period there were 152,877 cases of measles with 2,091 deaths, and 37,637 cases of pertussis with 2,384 deaths, which emphasizes the high mortality rate of whooping cough, over five times that of measles.

Reiche [16] reports the mortality statistics of the city of Hamburg for the 50-year period from 1872 to 1912, during which time there were 7,355 deaths out of 60,252 cases, with a percentage value of 12.2. From April, 1914, to August, 1920, there were sixty-nine deaths at the author's clinic in children under 6 years of age, forty-three of these deaths occurring in children under 3 years of age. Among these there were sixteen cases of tuberculosis in the 3 to 6 year age group, and four in the younger children, all of the latter showing miliary lesions. Eight patients had convulsions, but approximately 25 per cent. of the deaths were the results of a flare up of tuberculosis.

BLOOD

There were two articles reporting the blood findings during whooping cough. Hess [17] tabulates the leukocyte counts before and after the paroxysms in a large series of cases, and advances the theory that the paroxysm forces the white cells into the blood stream mechanically.

Bourne and Scott [18] report a case of whooping cough in a girl, aged 1 year and 9 months, in which the blood count on the sixteenth day of the disease reached 176,000. They raise the question whether there may not be some relationship between lymphatic leukemia and pertussis.

DIAGNOSIS

Modigliani and de Villa [19] were the first to report the use of intradermal inoculation of pertussis vaccine for diagnostic purposes. They used a loopful of a *Bacillus pertussis* culture in 1 c.c. of water containing a little of a 3 per cent. solution of toluene. They injected 0.1 c.c. of this mixture. In thirty-eight cases of pertussis they obtained positive results, in three patients during the catarrhal stage, even before paroxysms were noted. No reaction was noted in ten patients who had recovered from

16. Reiche, F.: Mortality of Whooping Cough, Med. Klin. **17**:33 (Jan. 9) 1921; Influenza Complicating Pertussis, München. med. Wchnschr. **67**:1352 (Nov. 12) 1920.

17. Hess, R.: Lymphocytosis of Pertussis, Ztschr. f. Kinderh. **27**:117, 1920.

18. Bourne, G., and Scott, J. M.: Whooping Cough With a Leukocytosis of 176,000, Brit. M. J. **1**:387 (March 11) 1922.

19. Modigliani, E., and de Villa, S.: Diagnosis by Intradermal Test, Pediatria **29**:337 (April 15) 1921.

pertussis. Other controls with bronchitis and coryza were uniformly negative. These investigators feel it offers a valuable means of early diagnosis.

Orgel [20] reports favorable results in a group of twenty-five cases with twenty-four positives and one negative. In fifteen controls the results were negative.

Hull and Nauss [21] find that the results are conflicting in a series of cases inoculated by the method of Modigliani, and do not feel that their results justify its further use.

Riesenfeld [22] also reported uncertain results, using various modified extracts of *Bacillus pertussis* intradermally.

Genoese [23] finds that the spinal fluid in six cases was negative and of no value diagnostically.

Niemann [24] feels that whooping cough is not a specific infection, and that other respiratory tract infections may be accompanied by typical whooping. He reports an epidemic of influenza in fifty-two children, nine of whom during the course of the disease showed the characteristic paroxysmal cough.

Gottlieb and Möller [2] find that the roentgen-ray appearance of the chest is diagnostic of whooping cough. In a considerable series of cases they comment on the uniformity of the picture. The diaphragm is low and its sides are extremely steep. The lung itself shows diffuse infiltration and is not cleared up after deep inspiration. The roentgen-ray picture of children with bronchitis and asthma are entirely dissimilar. The early diagnosis of whooping cough by means of this typical diaphragmatic picture can often be made before the paroxysmal cough appears. The left lower lobe seems to show the most frequent involvement, particularly at the point corresponding to the lower portion of the scapula. In the paroxysmal stage there is a hilum gland enlargement and peribronchial thickening as well.

PROPHYLAXIS

There are a number of articles on the prevention of whooping cough. These may be divided into those recommending isolation and those advocating vaccines. Of the former group Enklaar [25] suggests that by

20. Orgel, S. Z.: A Method for the Early Diagnosis of Pertussis. Preliminary Report, J. A. M. A. **79**:1508 (Oct. 28) 1922.

21. Hull, T. G., and Nauss, R. W.: Intracutaneous Reactions in Pertussis, J. A. M. A. **80**:1840-1841 (June 23) 1923.

22. Riesenfeld, Edwin A.: Intracutaneous Reactions in Pertussis, J. A. M. A. **80**:158 (Jan. 20) 1923.

23. Genoese, G.: Cerebrospinal Fluid in Pertussis, Policlinico (sez. prat.) **27**:291 (March 8) 1920.

24. Niemann, A.: Zum Keuchhustenproblem, Jahrb. f. Kinderh. **90**:77, 1919.

arranging school children in classes on the basis of their previous history of whooping cough and measles, effective prophylaxis among children may result by keeping those susceptible at home during an epidemic.

Stransky [26] reports an isolation system used in a hospital where small wards containing five beds each are arranged by glass partitions to enable the nurse on duty to see the patients. In such a ward of thirty beds only seven cases of whooping cough resulted during an epidemic, although no vaccines were used prophylactically for the other children.

The vaccine advocates range from those who report successful results in a small series of three or four cases to those reporting epidemics in large institutions.

Davies [27] reports an outbreak in the Syracuse State School for Mental Defectives. Of 177 girls there were thirty-three cases developing before prophylaxis was started. Of the 144 patients receiving prophylactic treatment only four developed the disease. Of the entire 177 girls only twenty had a previous history of having had the disease, so theoretically the others should have been susceptible.

Miller [28] reports successful use of prophylaxis vaccine in the public schools of Akron, Ohio, and states that sufficiently large doses will establish postive immunity, and check the spread of the disease. The failures are due to insufficient, timid dosage at too long intervals.

Bloom [29] in New Orleans is one of the most enthusiastic believers in prophylactic vaccination. He reports an outbreak of five cases in Saint Vincent's Foundling Asylum. Of the remaining 204 inmates who were given prophylactic vaccination, no new cases developed after several months. Three injections on alternate days were given, the dosage ranging from 0.25 c.c. for children under 6 months, to 1.25 c.c. for children over 2 years. He uses a vaccine made up of 5,000,000,000 *B. pertussis* and 3,500,000,000 influenza bacilli per cubic centimeter, prepared freshly once a week and used without any added preservative.

Spolverini [30] finds that prophylactic vaccination is effective in 93 per cent. of the cases.

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25. Enklaar, W. F.: Prevention of Whooping Cough, Nederlandsch Tijdschr. v. Geneesk. **1**:126 (Jan. 10) 1920.

26. Stransky, Eugen: Prophylaxis of Whooping Cough, Monatschr. f. Kinderh. **26**:410-414 (July) 1923.

27. Davies, G. R.: The Prophylactic and Therapeutic Value of Pertussis Vaccine, AM. J. DIS. CHILD. **23**:423 (May) 1922.

28. Miller, M. D.: Preliminary Report on Prophylaxis of Whooping Cough, Am. J. Pub. Health **11**:913 (Oct.) 1921.

29. Bloom, C. J.: Vaccine Therapy, Most Rational and Effective Method of Treating Whooping Cough, Arch. Pediat. **36**:1 (Jan.) 1919; Vaccine Therapy Most Rational and Effective Method of Preventing Whooping Cough in Public Institutions, New Orleans M. & S. J. **73**:426 (April) 1921.

30. Spolverini, L.: Vaccine Prophylaxis and Therapy, Policlinico (sez. prat.) **27**:1043 (Sept. 20) 1920.

Auricchio [31] also finds that large doses of freshly prepared pertussis vaccine prevent the development of the disease.

Hammes [32] has used vaccines for twelve years with successful results. Only temporary immunity is secured, however, and revaccination is by no means as effective.

Sajet and Van Gelderen [33] find that the general mortality rate of whooping cough has increased from 3.81 to 5.99 per cent., and that prophylactic vaccination is the only means of reducing this high rate, aimed particularly at young children by isolation, enforcing compulsory notification and hospitalizing cases.

McKibben [34] finds that fresh vaccine offers the most satisfactory means of reducing the incidence of the disease, particularly in infancy.

Debré [35] reports the successful prophylactic use of convalescent serum obtained from patients in the fourth week of the disease. He bases this therapy on the fact that this represents the period of maximum fixation of the complement. In forty cases, thirty-one patients were entirely protected, six ran a very mild course, and only three showed no apparent effect. The dosage is 3 c.c. given intramuscularly.

COMPLICATIONS

Kornmann [36] reports the finding of a hematuria during the early period of the disease in four cases out of five. He feels that this may be of diagnostic value.

Siegenbeek van Heukelorn [37] reports a post pertussis bronchiectasis in two cases in adults.

Morrison [38] presents an unusual complication of acute empyema during the course of the disease in a girl, aged 2½ years, who developed lobar pneumonia. Rib resection was done under local anesthesia, making an indirect opening and using the soft tissues as valve flaps, to overcome the spasmodic efforts of the cough.

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31. Auricchio, L.: Prophylaxis of Pertussis with Vaccines, Policlinico (sez. med.) **30**:25 (Jan.) 1923.

32. Hammes, F.: Die Kuhpocken impfug gegen Keuchhusten, Deutsch. med. Wchnschr. **47**:928 (Aug. 11) 1921.

33. Sajet, B. H., and Van Gelderen, J.: Voorkomen en Bestrijden Van Kinkhoest, Nederlandsch Tijdschr. v. Geneesk. **2**:1525 (Nov. 15) 1919.

34. McKibben, W. W.: Treatment of Whooping Cough in Private Practice, Boston M. & S. J. **189**:339-348 (Sept. 6) 1923.

35. Debré, R.: Preventive Serotherapy in Whooping Cough, Bull. Acad. de méd. **89**:348-351 (March 13) 1923.

36. Kornmann, F.: Ueber einen Befund von Erythrozten und Erythrozytenzylinden (liechte Hamaturie) in Harn bei Keuchhusten vor Ausbruch des spasmodischen Stadiums, München. med. Wchnschr. **62**:1422, 1915.

37. Siegenbeek van Heukelorn, J.: Changes in the Lungs Following Whooping Cough, Nederlandsch Tijdschr. v. Geneesk. **2**:855, 1920.

38. Morrison, W. R.: Acute Empyema Complicated by Whooping Cough, Boston M. & S. J. **184**:307 (March 24) 1921.

Nobécourt [39] discusses the relationship of tuberculosis and whooping cough. He feels that they may co-exist and each modify the course of the other, pertussis especially aggravating a latent tuberculosis, as about 5 per cent. of the whooping cough patients develop tuberculosis. When tuberculosis occurs during pertussis in infants, it is usually of the miliary form. In whooping cough positive tuberculin reactions are rare and it is often difficult to differentiate subacute bronchopneumonia from tuberculosis. Very careful hygiene with change of climate is usually indicated.

Galli [40] emphasizes the danger of diagnostic error through the temporary suppression of the specific allergy in tuberculous children, and recommends periodic examination to prevent a generalization of tuberculosis.

Jaureguy [41] reports three patients with fever, dyspnea and cyanosis complicated by tuberculosis, proved at necropsy, but in which the tuberculin reaction was negative.

Reiche [42] in several articles reports an outbreak of apparent whooping cough developing during an influenza epidemic. In twenty-nine cases with convulsions, whooping cough alone was found to be responsible for the convulsions in seventeen cases, while in the others some secondary factor entered. He believes that whooping cough complicated by convulsions is almost invariably fatal. He reports in detail the pathologic findings in twenty-six of the twenty-nine cases.

Hoffmann [43] reports nine cases of whooping cough with convulsions, with recovery following the use of 1 c.c. of 20 per cent. camphor liniment twice a day.

Canelli [44] found definite meningeal and cortical hemorrhagic foci at necropsy in two infants, aged 6 and 23 months. The infants died in coma and he feels that the hemorrhages were either due to asphyxia during the paroxysm or to the increased blood pressure present at that time, with a vessel wall weakened by the toxic action of the bacteria.

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39. Nobécourt, P.: Coqueluche et tuberculose, J. de méd. de Paris **41**:287, 1922.

40. Galli, P.: Tuberculin Allergy and Whooping Cough, Pediatria **31**:1091-1098 (Oct. 15) 1923.

41. Jaureguy, M. A.: Cyanosis in Whooping Cough, Rev. méd. del Uruguay **26**:207-212 (May) 1923.

42. Reiche, F.: Convulsions in Whooping Cough, Ztschr. f. Kinderh. **25**: 28 (May) 1920.

43. Hoffmann, W.: Convulsions in Whooping Cough, Schwiez. med. Wchnschr. **52**:438 (May 4) 1922.

44. Canelli, A. F.: Hemorrhagic Meningeal Complications with Whooping Cough, Pediatria **27**:351 (June) 1919.

Reh [45] reports a case of whooping cough with convulsions of an eclamptic type in a baby, aged 20 months. This was confirmed by histologic examination of the tissues. He feels that the convulsions and whooping cough are a mixture of tonic and clonic spasms.

Wernstedt [46] advances the theory that "spurious" whooping cough may occur in spasmophilic babies during ordinary nasopharyngeal infections. He cites an outbreak of this character in a childrens' institution, and further supports his theory by citing the case of a baby, aged 5 months, in which the "whoop" could be made to appear or disappear simply by alteration of the diet.

Blühdorn [47] feels that the recognition of latent spasmophilia in whooping cough is important, for the early administering of cod liver oil and calcium and the elimination of milk from the diet will often give relief.

Cantilena [48] also emphasizes the importance of the relationship of whooping cough and tetany, particularly in artificially fed infants.

Cohn [49] reports an epidemic of combined measles and whooping cough in which the blood picture offered considerable difficulty.

Schmitt [50] reports a case of transient blindness in a child, aged 2½ years, with a severe case of whooping cough, complicated by convulsions and some temporary muscular paralysis.

TREATMENT

The articles on treatment can be divided into five main groups, with a few other miscellaneous forms of therapy. These groups consist first, of a varied assortment of drugs used systemically; second, of the local applications of various drugs to the nasopharynx and larynx; third, a group consisting of the intramuscular injection of the ether as originally suggested by Audrain in 1914; fourth, a group composed of a number of reports of vaccine therapy and finally the fifth group, including the various forms of applied physiotherapy, using diathermy, the ultraviolet lamp and the roentgen ray. Of the miscellaneous forms of treatment the following are of sufficient interest to mention.

45. Reh, T.: Eclampsie pseudo-tetanique au decourse de la coqueluche, Bull. Soc. de pédiat. de Paris **20**:261, 1922.

46. Wernstedt, W.: Spurious Whooping Cough in Spasmophilia, Hygiea **82**:559 (Sept. 16) 1920.

47. Blühdorn, K.: Whooping Cough Convulsions and Spasmophilia, Ztschr. f. Kinderh. **26**:251 (Sept.) 1920.

48. Cantilena, A.: Whooping Cough and Breast Feeding, Pediatria **31**:555-560 (May 15) 1923.

49. Cohn, E.: Blood Picture in Measles Plus Whooping Cough, Deutsch. med. Wchnschr. **47**:1357 (Nov. 10) 1921.

50. Schmitt, W.: Transient Blindness in Whooping Cough, Klin. Wchnschr. **2**:1413 (July 23) 1923.

Luttinger [51] recommends the use of a special abdominal binder made of a long staple cotton, especially woven on the bias to secure the maximum elasticity. This is kept in stock in different sizes by several medical supply houses.

Schroeder [52] has tried the alcoholic injection of the superior laryngeal nerves in eleven cases, in which improvement was noted in two. He feels that the method is theoretically good, but the dosage used was inadequate.

Biemann [53] attempted similar nerve blocking with a procain alcohol mixture in twelve cases, with only one success. He warns against the the use of this method as it seems to irritate the sympathetic nervous system, and two of his patients developed left sympathetic paralysis.

Döbeli [54] argues that by checking the expectoration the paroxysms will be diminished. He handles his patients chiefly by dietary measures aimed at reducing the uric acid production and keeping the bowels open. He notes that the disease usually is seen in over fed children with excitable central nervous symptoms, and by these hygienic precautions the general condition is improved.

DRUG THERAPY

There have been a great variety of drugs used in the treatment of the disease, both systemically and locally. Of the first group the following are the most important.

Macht [55] reports a study of 115 cases, using a 20 per cent. solution of benzyl benzoate by mouth, of which 90 per cent. showed definite improvement. He does not claim that the treatment is anything but palliative. He feels that the satisfactory results are due to the fact that the drug has first an antispasmodic action on the bronchial spasm; second, it is a sedative of striated muscle; third, it is anesthetic to the larynx, thus diminishing the irritability of the mucosa; fourth, it is an expectorant; fifth, it is a definite sedative to the respiratory center and sixth, it may have some antiseptic action.

McMurray [56] confirms the favorable reports of the treatment of whooping cough by benzyl benzoate in a few cases.

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51. Luttinger, P.: Vaccine Therapy as Routine Treatment in Whooping Cough, New York M. J. **109**:322 (Feb. 22) 1919.

52. Schroeder, C. B.: Blocking Nerve in Whooping Cough, Klin. Wchnschr. **2**:1545-1548 (Aug. 13) 1923; Treatment of Whooping Cough, Med. Klin. **19**: 974-976 (July 15) 1923.

53. Biemann: Novokian-Alkohol-Injektionen bei Pertussis, Deutsche Med. Wchnschr. **48**:508, 1922.

54. Döbeli, E.: Treatment of Whooping Cough, Schweiz. med. Wchnschr. **51**:224 (March 10) 1921.

55. Macht, D. I.: Experimental and Clinical Therapeutic Study of Whooping Cough, Bull. Johns Hopkins Hosp. **31**:236 (July) 1920.

56. McMurray, T. E.: Benzyl Benzoate Treatment of Whooping Cough, New York M. J. **112**:122 (July 24) 1920.

Hirsch [57] in five years has treated 370 cases with diothymin, which is a shot-gun mixture made up chiefly of dionin and thymien. The dosage varies with the age of the patient. His patients are kept in bed for a week and given the drug at three hour intervals. The average length of time for cure is one month.

Carriére,[58] since 1919, has adopted a routine treatment which he feels is very successful. In 500 selected cases he has secured a cure in from eight to ten days on the average, the longest being three weeks. The mortality has been 1.5 per cent., out of 390 cases closely followed, as compared with a mortality rate of from 13 to 29 per cent. in his series from 1900 to 1905. The method of treatment has first of all been cubicle isolation of each case. Second, in covering the bed with a tent of muslin on which a tampon of eucalyptol is placed three or four times a day. Third, irrigation of the nasal fossae three times a day by syringing with a 1 per cent. resorcinal mixture containing 5 per cent. borated glycerin, and fourth, daily intramuscular injection of an oil of cajuput preparation.

Schmidt [59] used phenobarbital in doses of 0.5 gm. a day, even in young infants. The results were not as satisfactory as expected, but he feels that the treatment is of definite value.

Hall [60] reports the finding of a pencillium in the stomach washings of eight patients with whooping cough. He suggests that the cause of the disease may be a fungus infection of the stomach, as his patients were cured in from three to six days by gastric lavage, twice a day, using 2 quarts of water containing 20 drops of compound solution of cresol, or 4 drams of sodium bicarbonate.

Wilhelm [61] feels that there is no specific therapy for whooping cough, and that the disease must be treated symptomatically. Gastric lavage proved to be of great benefit, particularly in neuropathic children.

Alexander [62] recommends the use of an ozone apparatus in the rooms of the patients with whooping cough. He reports a single case showing great benefit by this treatment.

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57. Hirsch: Die Behandlung des keuchhustens mit diothymin, Med. Klin. **18**:1346 (Oct. 15) 1922.

58. Carriére, G.: Treatment of Whooping Cough, Bull. Acad. de méd. **88**: 30 (July 11) 1922.

59. Schmidt, M.: Treatment of Whooping Cough with Phenolbarbital, Med. Klin. **19**:314 (March 11) 1923.

60. Hall, T. P.: Cause of Whooping Cough: Stomach Lavage Used as a Therapeutic Measure, New York M. J. **116**:158 (Aug. 2) 1922.

61. Wilhelm, M.: Gastric Lavage in Whooping Cough, Therap. Halb-Monatsch. **34**:443 (Aug. 15) 1920.

62. Alexander, F. W.: Treatment of Whooping Cough by Ozone, Lancet **1**: 199, 1922.

Von Starck [63] has used various forms of quinin preparations with good results in a number of cases. He recommends intensive treatment of children with spasmophilia, tuberculosis, or spasm of glottis, as they are particularly susceptible to the disease.

Heinz and Schottenheim [64] report the use of a thyme preparation in the treatment of pertussis, the dosage being from 1 to 4 drops.

Catel [65] following Rietschel has treated twelve patients with a proprietary preparation stated to be iodo-sulphon-oxybenzol pyridin, and agrees with Gottlieb and Möller [2] that it is of very little value therapeutically. Schmuckler [66] has had satisfactory results in sixty-two patients treated by a 5 per cent. solution of this preparation given intramuscularly in doses of from 3 to 5 c.c. on alternate days.

LOCAL DRUG TREATMENT

Local treatment by various drugs is reported by several men.

Akkersdijk [67] and Yankauer [68] both report the local use of a 4 per cent. solution of antipyrin to the larynx daily. The basis of this treatment is that the only visible pathology seems limited to the larynx.

Ochsenius [69] and Lederer [70] both report the use of 2 per cent. silver nitrate locally, the former treating twice a day, the latter once every two days. Ochsenius has treated 870 cases in the last twelve years, with only eight deaths. Lederer reports the successful treatment of a small series of twenty-five cases and attributes much of its effect to suggestion.

Meyer-Houssell [71] believes that the initial irritation of whooping cough is in the posterior nasal cavity and treats this area energetically with either an iodin-phenol-glycerin mixture (Bradt) or with 2 per cent. silver nitrate (Ochsenius).

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63. Von Starck: Zur Chinintherapie des Keuchhustens, München. med. Wchnschr. **67**:455, 1920.

64. Heinz, R. A. F., and Schottenheim: Thymipin in Treatment of Pertussis, München. med. Wchnschr. **67**:771 (July 2) 1920.

65. Catel, W.: Deutsch. med. Wchnschr. **49**:762 (June 8) 1923.

66. Schmuckler, A.: Treatment of Whooping Cough, Deutsch. med. Wchnschr. **49**:484 (April 13) 1923.

67. Akkersdijk, W.: Behandlung van Kinkhoest met antipurine, Nederl. Tijdschr. v. Geneesk. **2**:653, 1915.

68. Yankauer, S.: Symposium on Border-Line Diseases, Laryngoscope **31**:101 (Feb.) 1921.

69. Ochsenius, K.: Treatment of Whooping Cough, Deutsch. med. Wchnschr. **49**:988-989 (July 27) 1923.

70. Lederer, R.: Treatment of Pertussis with Silver Nitrate, Wien. klin. Wchnschr. **33**:1049 (Nov. 25) 1920.

71. Meyer-Houssell, O.: Aktive Keuchhustenbehandlung, Deutsch. med. Wchnschr. **47**:996, 1921.

Genoese [72] has used a 25 per cent. solution of magnesium sulphate intramuscularly in four cases complicated by spasmophilia with satisfactory results.

Martinez-Vargas [73] uses a preparation stated to be potassium sulphonapholate locally in the pharynx.

Parry [74] recommends a preparation of powdered lactic acid bacilli, which is insufflated three or four times a day. He has used it successfully in thirty cases, the "whoop" disappearing in from two to eight days. He attributes this to a protein reaction, which overcomes the toxemia of the *Bacillus pertussis.*

Macleod,[75] on the basis of Barlow's report of fifty cases, has used a 10 per cent. resorcinol solution in a mixture of 1 part glycerin and 17 parts water. He brushes out the pharynx hourly during forced respiration with the tongue extended and the mouth wide open. This he feels does two things. It removes the mucus and reduces the irritability of the pharyngeal reflex. The results are about as satisfactory using salt solution, and he feels that it is the mechanical and psychic effect, rather than any inherent effect of the drug.

Sobel [76] recommends a mechanical method for controlling the paroxysms. This consists of pulling the lower jaw downward and forward with the thumbs. It is more successful in the older children.

Camescasse [77] recommends simply a change of climate for the treatment of the disease, and the establishment of hospitals in the country for the treatment of whooping cough.

ETHER

Audrain,[78] in 1914, first introduced the treatment of whooping cough by intramuscular injections of ether. In 1920 he reports further work by this method. In his conclusion he states that there is a definite antibactericidal action on the part of the ether, when either given subcutaneously or by inhalation. He reports cures in from two to ten days

72. Genoese, G.: Magnesium Sulphate in Spasmophilia and Whooping Cough, Gazz. d. osp. **40**:891 (Oct. 16) 1919.

73. Martinez-Vargas: Syncope and Apparent Death in Whooping Cough, Arch. espan. de pediat. **5**:257 (May) 1921.

74. Parry, F.: Note on Treatment with Lactic Acid Bacilli Preparations, Lancet **1**:78 (Jan. 13) 1923.

75. Macleod, N.: A Problem in the Treatment of Whooping Cough, Lancet **1**:254 (Feb. 15) 1919.

76. Sobel, J.: Whooping Cough; a Simple Method to Control the Paroxysms, Arch. Pediat. **35**:182, 1918.

77. Camescasse, J.: De l'heure opportune pour changer d'air des coquelucheux; de l'utilisation par "l'Assistance publique" de cette notion, Arch. de méd. d. enf. **22**:466 (Sept.) 1919.

78. Audrain, J.: Le traitement de la coqueluche par l'ether, Bull. et mém. Soc. méd. d. hôp. de Paris **44**:795 (June 11) 1920; ibid., Médecine **1**:694 (Aug. 20) 1920.

using from 1 to 2 c.c. doses once to three times a day. In patients showing adenoidal pathology the treatment is not effective, and accordingly offers an excellent means of differential diagnosis of doubtful cases of cough.

Cheinisse [79] supports the theory of Audrain in a series of cases which he reports.

Veronese [80] treated ten patients with intramuscular injection of ether with improvement in five and cures in two. No changes were noted in the other three patients.

D'Aroma [81] finds ether the most useful drug in the treatment of whooping cough. In several patients he has used as high a dose as 6 c.c. daily.

Genoese [82] reports fifty patients benefited by the treatment with intramuscular injections of ether. He recommends it as practical, harmless, inexpensive, and the most valuable addition to our armamentarium since the introduction of vaccine.

Lassablière,[83] Klotz [84] and Veltoni [85] all find that the intramuscular injection of ether gives more satisfactory results than any other drugs. It usually takes from six to ten injections and improvement is noted after the second or third injection.

Reim [86] has treated thirty-seven cases using 1 c.c. of ether combined with camphor. After from five to six injections daily the paroxysms were reduced from twenty and twenty-four to six and eight. No bad results were seen even in infants. One patient had a slight subcutaneous abscess.

Magni [87] has employed ether in the treatment of thirty-five children, with seventeen cures, eight patients showing improvement and nine showing no change. He thinks the mechanism of its action is a combination of its tonic, antispasmodic, febrifugal and antitoxic effect. The

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79. Cheinisse, L.: Ether Injections in Whooping Cough, Presse méd. **28:** 526 (July 31) 1920.

80. Veronese, L. D.: Intramuscular Injection of Ether in Treatment of Pertussis, Gazz. d. osp. **42:**332 (April 7) 1921.

81. D'Aroma, A.: Ether Treatment of Whooping Cough, Policlinico (sez. prat.) **28:**788 (June 16) 1921.

82. Genoese, G.: Terapia della pertosse con l'etere, Policlinico (sez. prat.) **29:**1105 (Aug. 21) 1922; ibid. **28:**788 (June 6) 1921.

83. Lassablière, P.: Treatment of Whooping Cough, Médecine **2:**883 (Aug.) 1921.

84. Klotz: Zur Behandlung des Keuchhustens, München. med. Wchnschr. **69:**1431 (Oct. 6) 1922.

85. Veltoni: Die Behandlung des Keuchhustens, München. med. Wchnschr. **69:**1012 (July 7) 1922.

86. Reim: Zur Behandlung des Keuchhustens mit Aetherinjektionen, Med. Klin. **18:**698 (May 28) 1922.

87. Magni, Z. L.: Le iniezioni intramuscolari di etere nella cure della tosse convulsa, Riv. di clin. pediat. **20:**285, 1922.

method of elimination in a large part through the lungs allows the drug to reach the most minute recesses and exert some modifying destructive effect on the bacteria. Another possible method of action may be through stimulation of phagocytosis or raising the alexin content in the blood.

Graeser [88] reports in detail the treatment of twenty-one patients with ether, ranging in age from 2 months to 3 years. There were three complete cures, fourteen patients showing improvement and two which did not show any effect. The average duration of the disease was about a week after treatment, with marked improvement after the second and third day.

Bedö [89] recommends the use of ether only in the severe cases as it is extremely painful to young children. He had noted the value of anesthesia in two surgical patients who had whooping cough and to whom a general anesthetic was administered, previous to the reported work on the value of intramuscular ether.

Panayotatou [90] reports an outbreak of twenty-five cases of whooping cough treated by 2 c.c. of ether intramuscularly on alternate days. The average duration was twelve days.

Mason [91] reports the results of intramuscular injection of ether in twenty-six cases. Eleven patients stopped coughing after the eighth injection, six were improved and four showed no change. Four patients were given the ether combined with olive oil rectally every six hours. Two of these stopped coughing promptly and the other two showed no change.

VACCINES

Bloom [29] reviews the literature of the last ten years on vaccine therapy in whooping cough, bringing out the fact that most of the favorable results have been in the hands of those men using massive doses of a freshly prepared vaccine of known high antibody productivity. Of his own work he concludes that vaccine therapy is effective, both prophylactically and therapeutically. It minimizes loss in weight, reduces the duration of the disease, decreases the severity of the paroxysms, foreshadows complications, limits the mortality, and is unattended by anaphylaxis. He reports the use of vaccine in three series, including

88. Graeser, F.: Treatment of Whooping Cough with Ether, Deutsch. med. Wchnschr. **49**:551 (April 27) 1923.

89. Bedö, J.: Ether in Treatment of Whooping Cough, Ztschr. f. Kinderh. **35**:325-326 (June) 1923.

90. Panayotatou, A.: Twenty-Five Cases of Whooping Cough Treated with Injections of Ether, Brit. J. Child. Dis. **20**:151-153 (July-Sept.) 1923.

91. Mason, C. C.: Treatment of Pertussis with Intramuscular Injection of Ether, J. A. M. A. **81**:2114-2115 (Dec. 22) 1923.

seventy-three cases. He uses a vaccine composed of the following: Bordet-Gengou bacillus, 1,000,000,000; *Staphylococcus pyogenes-aureus,* 500,000,000,000; *Micrococcus catarrhalis,* 40,000,000,000; Influenza bacillus, 160,000,000,000; *Streptococcus pyogenes-aureus,* 100,000,000,000. In the two series using this vaccine, the duration after treatment was instituted was reduced to between ten and fifteen days.

Luttinger [92] reports 238 cases (fifty-one prophylactic) following his report of a year before of 7,500 cases. Out of the fifty-one prophylactic patients only two developed a whoop and seven a cough. Of the 187 curative cases the average duration was sixteen days after the first injection. He gives from three to six doses on alternate days, doubling the dose each time, with an initial dose of one-half billion and reaching as high as 16 billion.

Reynolds [93] reports fifty-one cases treated by vaccine, twenty-one with a mixed vaccine, and thirty with a straight pertussis vaccine. He concludes that there is some virtue in unmixed vaccine, but more with mixed vaccine, and that vaccine therapy offers the best methods of treatment with our present knowledge.

Rogers [94] reviews the literature concisely and reports eleven cases benefited by vaccine. He concludes that there is enough evidence of a conclusive nature piling up to warrant the vaccine treatment of whooping cough as the most rational procedure the profession as yet has at hand.

Cammarata [95] reports ten cases in which vaccine therapy was used with some benefit.

Freeman [96] reports the use of a fresh Lederle vaccine in sixteen cases, with four cures, seven patients showing improvement, and five not being affected. The dosage was one-half million to two million of organisms. He thinks that it should be used much more extensively and advises the use of fresh vaccine.

Auricchio [97] reports the results of 196 cases using Caronia's vaccine, which is made of the autolysate of a forty-eight hour culture of *Bacillus pertussis,* with 5 per cent. phenol and 0.85 per cent. sodium chlorid added. He gives a dose of 4 billion organisms on alternate days.

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92. Luttinger, Paul: An Abdominal Bandage for Patients with Whooping Cough, J. A. M. A. **78**:1536 (May 20) 1922.

93. Reynolds, H. S.: Vaccine Therapy in Fifty-One Cases of Pertussis, Arch. Pediat. **36**:290 (May) 1919.

94. Rogers, F. N.: The Present Status of Vaccine Therapy in Whooping Cough, Tr. New Hampshire M. Soc. **128**:137, 1919.

95. Cammarata, A.: Vaccine Therapy of Whooping Cough, Policlinico (sez. prat.) **27**:1442 (Dec. 13) 1920.

96. Freeman, R. D.: Use of Fresh Vaccines in Whooping Cough, Med. Rec. **98**:762 (Nov. 6) 1920.

97. Auricchio, L.: Vaccine Therapy of Whooping Cough, Pediatria **29**: 1009 (Nov. 15) 1921.

Of the 196 cases, 67.8 per cent. were cured, 26 per cent. improved and only 6 per cent. failed to show relief. In these latter fourteen cases the disease was well advanced. In twenty-four control cases treated by ether no results were obtained in 87.5 per cent.

Bruce [98] recommends the use of vaccines and benzyl benzoate in the treatment of whooping cough.

Davison,[99] in a special article discusses the value of vaccines in the treatment of whooping cough. He states that in summing up the prolific and somewhat contradictory literature on the subject it may be concluded that injections of Bordet-Gengou vaccine may have a slight, though unreliable prophylactic effect, and that therapeutic inoculations are of practically no value.

Rewalt [100] finds that the vaccine treatment is the most valuable weapon we have, using large doses at short intervals. He reports 130 patients, of which seventy-nine showed marked improvement, forty-five showed definite benefit, and only six in which no improvement was noted; he uses a dose of 250,000,000 up to 2,000,000,000 bacteria per cubic centimeter, employing the straight pertussis vaccine, except in cases complicated by pneumonia, in which a mixed vaccine is used.

Appel and Bloom [101] report the use of vaccine in an outbreak in the Israel Orphan Asylum in New York. In one house where thirty-one children lived, fourteen of them developed the disease. They were given four doses of mixed vaccine and all cured within twelve days. The others were given prophylactic vaccine, and no new cases resulted. Forty-seven other children were sent away to another institution where they became exposed to pertussis. Twenty-seven of these developed whooping cough before vaccination was begun. The others were protected by prophylactic vaccine. They used a mixed vaccine containing the following: *Bacillus pertussis,* 1,000,000,000; *Micrococcus catarrhalis,* 200,000,000; *Staphylococcus aureus,* 200,000,000; *Staphylococcus pyogenes,* 200,000,000; *Bacillus influenzae,* 400,000,000; Pneumococcus Types 1, 2, 3 and 4, 50,000,000 each. The dosage was graded from 0.5 to 2 cm. given on alterante days for four doses. He reviews the literature on whooping cough commenting on the fact that in the last ten years in the United States there have been over 100,000 deaths from whooping cough, and emphasizes the importance of any treatment aimed at combating a disease with such a mortality.

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98. Bruce, J. W.: Recent Development in Contagious Diseases, Kentucky M. J. **19**:5 (Jan.) 1921.

99. Davison, W. C.: Pertussis Vaccine, J. A. M. A. **76**:242 (Jan. 22) 1921.

100. Rewalt, R. K.: Vaccine Treatment of Pertussis, Pennsylvania M. J. **24**:404 (March) 1921.

101. Appel, H. N., and Bloom, O. I.: Arch. Pediat. **39**:145 (March) 1922.

Gonzalez-Alvarez [102] reports the use of "Ferran's antialfa vaccine" in 242 new cases. Seventy-five patients were cured, sixty-nine showed marked improvement, in fifty no change was noted and in eight the treatment was suspended because of a complicating pneumonia. From three to five injections were usually required and a cure in two weeks on the average was affected. He states that in Alciria, Spain, 30,000 vaccinations have been made with this vaccine. Ferran believes that it attacks not only the alfa organisms, but other symbiotic bacteria. Tuberculosis did not occur in Ferran's series.

Paterson and Smellie [103] record their observations in 804 cases seen during 1921. The seasonal instance showed its maximum in June (109 cases) and the lowest figure in November (twenty-five cases). The results in fifty-eight cases treated by vaccines showed no appreciable benefit, and the authors deprecate the vaccine treatment of the disease, and do not believe that the results justify its further use.

Wolff [104] reviews the literature and the experimental evidence in favor of a specific vaccine therapy for whooping cough. He recommends the use of large doses of fresh vaccine.

Hayano [105] recommends the use of vaccines therapeutically as he found 94.8 per cent. improvement in 159 cases. It is a very technical article with numerous tables of the various precipitation and agglutination reactions, following which he concludes that they are of little value diagnostically.

Stern [106] obtained definite diminution of the intensity and duration of the disease by the injection of 20 c.c. of the serum of calves vaccinated with cowpox, following the method of Violi. The curative effect of the smallpox vaccination of children with whooping cough had already been shown by Jenner.

Reiche [107] treated thirteen boys and six girls in varying stages of the disease with Violi's serum. The dosage was from 15 to 20 c.c. intramuscularly at ten day intervals. No results were noted either in

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102. Gonzalez-Alvarez, M.: Vaccine Therapy of Whooping Cough, Arch. espan. de pediat. **6**:193 (April) 1922.

103. Paterson, D., and Smellie, J. M.: Value of Vaccines in Treatment of Whooping Cough, Brit. M. J. **1**:713 (May 6) 1922.

104. Wolff, L. K.: Vaccine Therapy of Whooping Cough, Nederlandsch Tijdschr. v. Geneesk. **2**:2174 (Nov. 11) 1922.

105. Hayano, M.: A Classification of B. pertussis; Vaccine Therapy and Prophylaxis for Whooping Cough, Japan M. World **3**:104-105 (May) 1923.

106. Stern, G.: Vaccine Therapy for Whooping Cough, Monatschr. f. Kinderh. **22**:190 (Nov.) 1921.

107. Reiche, F.: Zure Behandlung des Keuchhustens nach Violi, Med. Klin. **17**:1573 (Dec. 25) 1921; Mortality of Whooping Cough, Med. Klin. **17**:33 (Jan. 9), 1921; Influenza Complicating Pertussis, München. med. Wchnschr. **67**:1352 (Nov. 12) 1920.

reducing the number or severity of the paroxysms. The only change noted clinically was a decrease of the characteristic leukocytosis, but even this change was not constant.

Lesné and Petot [108] use fresh noncitrated blood obtained in the fourth week of the disease and injected intramuscularly daily. They do not find any effect on the paroxysms, but find that it acts almost as a specific in overcoming complicating pneumonias.

Méry and Girard [109] report a case of a baby, aged 2 months, on whom convalescent serum was used, with marked benefit. The baby was infected from the mother. The disease began the end of January. In April, 20 c.c. of whole citrated blood from the mother was introduced intramuscularly. Two days later 10 c.c. more of the blood, which had been preserved on ice during the interval, was administered. Complete cessation of symptoms resulted, except for one or two mild attacks a day.

PHYSIOTHERAPY

Kleinschmidt [110] reports the use of diathermy in the treatment of whooping cough. Two thin lead electrodes covered with flannel soaked in saline solution were used. The strength of the current varied from 3 to 5 amperes depending on the age of the child. This was administered from five to fifteen minutes twice a day. He obtained excellent results following ten or twelve treatments.

Gaerntner [111] treated his own child with radiation of the pharynx by concentrated sunlight. The attacks were decreased in number and intensity. As soon as his second child developed symptoms he treated it in the same manner, the case was almost aborted, and the child became immune, as evidenced by the complement fixation. The method employed is to place the patient with the back toward the sun so that the sun's rays will fall on a mirror of a laryngeal reflector. An exposure of from ten to twenty seconds is given, repeating several times in succession, twice a day.

Schotten [112] recommends the use of ultraviolet rays in the treatment of the disease. He exposes the chest for five minutes. After five days a second exposure, regulated according to the skin reaction, is given

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108. Lesné and Petot: Serotherapy of Whooping Cough, Bull. et mém. Soc. méd. d. hôp. de Paris **47**:323-324 (March 2) 1923.

109. Méry, H., and Girard, L.: Hemotherapy in Whooping Cough, Bull. et mém. Soc. méd. d. hôp. de Paris **47**:243-244 (Feb. 16) 1923.

110. Kleinschmidt, K.: Treatment of Pertussis by Diathermy, Med. Klinik **16**:1206 (Nov. 21) 1920.

111. Gaerntner, G.: Ein Vorschlag zur Therapie des Keuchhustens, Wien. med. Wchnschr. **69**:2033, 1919.

112. Schotten, F.: Treatment of Whooping Cough with Ultraviolet Rays, Deutsch. med. Wchnschr. **49**:1157 (Aug. 31) 1923.

increasing the duration by from three to five minutes and alternating front and back. The paroxysms are reduced in severity and number, but the duration of the disease as a whole is not shortened.

Bowditch [113] in two recent articles reports the use of the roentgen ray in the treatment of the disease. His preliminary report with Leonard covered twenty-six cases, in which marked benefit was noted. The subsequent report summarizes the results in 300 cases. About 80 per cent. of the cases showed definite improvement, while in from 15 to 20 per cent. of the cases a cure was obtained following one or two treatments. The method of treatment was to expose the chest first anteriorly and then posteriorly for three times on alternate days, using a 6 inch back up spark, a 1 mm. aluminum filter and the tube placed 24 inches (60 cm.) from the patient. The dosage ranged from 8 to 20 milliampere minutes. An interval of a week then ensued and a second series of treatment was given to those patients in which improvement was not entirely satisfactory. Very few patients received more than four treatments. Roentgen-ray plates were taken on the first and last treatments and blood studies made at frequent intervals.

Kingston and Faber [114] likewise, following Bowditch's technic, recommend the use of the roentgen-ray treatment of whooping cough. They comment particularly on the cessation of vomiting as being an almost constant effect of the roentgen ray. Their statistics are comparable to those of Bowditch. They state that: "it is our belief that the roentgen-ray treatment is at present the most promising therapeutic measure which we possess."

Struthers [115] from Montreal likewise reports forty-five cases treated at the Montreal General Hospital by the roentgen ray. They ranged in age from 3 months to 45 years. In about 60 per cent. of the cases definite improvement was noted within a short time. He has modified Bowditch's technic by using a large dose instead of the small repeated dose. He says: "I know of no other method of treatment which gives as good results."

McKibben,[34] in his article previously cited, comments in conclusion that the best form of therapy in the treatment of whooping cough seems to be a combination of the roentgen ray supplemented by large doses of freshly prepared vaccine.

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113. Bowditch, H. I., and Leonard, Ralph D.: Preliminary Report of the Treatment of Whooping Cough by Means of the Roentgen Ray, Boston M. & S. J. **188**:312 (March 8) 1923; Bowditch, Henry I.: Further Notes on the Treatment of Pertussis by the Roentgen Ray, J. A. M. A. **82**:1422 (May 3) 1924.

114. Kingston, J. J., and Faber, H. K.: Notes on Roentgen-Ray Treatment of Whooping Cough, California State J. Med. **21**:429-430 (Oct.) 1923.

115. Struthers, R. R.: Roentgen-Ray Treatment of Pertussis, Canad. M. A. J. **14**:141-142 (Feb.) 1924.

SUMMARY

The literature of whooping cough during the last five years brings out certain very interesting points. There is no uniformity of opinion as to the value of any one method of treatment, as evidenced by the number of remedies reported. It seems generally conceded that drug therapy is at the best only symptomatic and palliative. A considerable diversity of opinion exists as to the value of vaccines. The consensus of opinion would seem to suggest that the prophylactic use of vaccine may be expected to yield very satisfactory results, when used in sufficient dosage. The therapeutic value of vaccines on the other hand seems to be open to considerable doubt. The only men reporting success in the use of vaccine therapeutically are those who use exceedingly large doses of a freshly prepared vaccine given at frequent intervals, either daily or on alternate days. The various forms of physiotherapy seem to offer more than the other methods of treatment. Perhaps the ideal treatment at the present time may be that suggested by McKibben in the last article quoted, in which he states that the combination of the roentgen ray and adequate doses of vaccine give the most beneficial results. The disease is one which still remains to be solved by further experimental work. With its etiology conceded by all investigators to be due to the *Bacillus pertussis,* it seems as if the day should not be far away when a specific therapy should be developed, as in the case of diphtheria, scarlet fever and other infectious diseases.

Abstracts from Current Literature

Anatomy and Physiology

Observations on the Origin of Urinary Ammonia. Robert F. Loeb, Dana W. Atchley and Ethel M. Benedict, J. Biol. Chem. **60**:491, 1924.

Determinations of the ammonia content of blood taken from an artery, the renal vein and the vena cava were made on dogs and rabbits. In dogs the renal vein showed a higher content of ammonia than arterial or other venous blood. In rabbits the blood from the renal vein has the same percentage of ammonia as the blood from the aorta. The rabbit excretes essentially no ammonia in the urine. The results of the experiments indicate that urinary ammonia is formed in the kidney.

Hydrogen-Ion Concentration in the Human Duodenum. H. V. Hume, W. Denis, D. N. Silverman and E. L. Irwin, J. Biol. Chem. **60**:633, 1924.

A patient had been shot in the abdomen and a duodenal fistula resulted. An electrode was inserted through the opening and the hydrogen-ion concentration of the duodenal contents determined. The average of 182 determinations was p_H 7.02 (range from p_H 5.90 to 8.23). No definite variation following food was observed.

Uric Acid Excretion. Alfred E. Koehler, J. Biol. Chem. **60**:721, 1924.

Uric acid in doses of 12 mg. per kilogram of body weight was given intravenously to normal persons and patients with renal impairment. The uric acid in the blood was increased two hours after the injection from 25 to 75 per cent. in normal persons, but in nephritic patients often over 100 per cent. Only about 50 per cent. of the injected uric acid was excreted in the urine. The author suggests that the procedure affords a possible method of determining renal function.

Higgins, Cincinnati.

The Physiology and Pathology of the Small Intestine. A. Adam, Ztschr. f. Kinderh. **38**:378 (Part I) and 386 (Part II), 1924.

Studies were made of the intestinal peristalsis, strength and duration, in vitro under various conditions. The acid and alkaline reactions produced had little influence on the peristalsis of normal small intestines. The mode of feeding and the volume of the food, and the emptying time of the stomach influence the peristaltic activity of the small intestine of infants with diarrhea. In severe diarrhea, if all fluid is given extra-orally with small amounts of food by mouth the stools approach normal.

Pulse Frequency of the Fetus, Infant and Small Child. J. John and B. Schick, Ztschr. f. Kinderh. **38**:216, 1924.

Studies were made to determine the relation between the pulse rate and the sitting height. In the young children the "sitting height pulse" (pulse rate in as many seconds as the sitting height in centimeters) was 100. With a growing sitting height the "sitting height pulse" remains the same, the pulse rate per minute decreases. The fetal pulse rate remains the same in the second half of pregnancy. In 531 fetuses the minute pulse rate averaged 134. The "sitting height pulse" gradually increases. Two "sitting height pulse" factors

are to be added to each centimeter of growth of the sitting height. Thus it is impossible to determine the size of the fetus from the pulse rate per minute. The "sitting height pulse" of the fetus, the new-born, and the infant is smaller than that of the older child. In the fetus with a sitting height of 33 cm. the sitting height pulse was 74. After birth it was 65, in the sixth year 100. These factors indicate that the heart of the fetus, new-born infant and young children has a larger capacity in proportion to the circulation than it has in older children. The heart is also anatomically larger in proportion to the body weight.

PETERMAN, Rochester, Minn.

STUDIES ON THE SALIVARY AMYLASE OF THE SUCKLING IN THE EARLY MONTHS OF LIFE. CESARE COCCHI, Riv. di clin. Pediat. **22**:449, 1924.

Cocchi finds the saliva of newly born infants and sucklings strongly amylolytic. The activity varies over wide limits, is independent of the food and is not sensibly altered during disease. He finds, in fresh saliva, a thermostable substance more active in adults than in children, which is capable of raising the amylolytic power of fresh saliva and especially the power to digest cooked starch in fresh vegetable tissue.

SHIPLEY, Baltimore.

Pathology, Bacteriology and Biochemistry

LEAD STUDIES. III. THE EFFECTS OF LEAD ON RED BLOOD CELLS. PART 1. CHANGES IN HEMOLYSIS. JOSEPH C. AUB, PAUL REZNIKOFF and DOROTHEA E. SMITH, J. Exper. Med. **40**:151, 1924.

Washed red blood cells exposed to small quantities of lead chlorid develop increased resistance to hypotonic salt solution, so that complete hemolysis occurs only when the strength of the salt solution is reduced to 0.05 per cent.

This reaction is quantitative and varies with the concentration of lead used.

The treatment with lead appears to shorten the life of the blood cells as is demonstrated by more rapid appearance of hemolysis than normal when the "leaded" cells stand in Ringer solution of any dilution.

These same phenomena may be noted in vivo in rabbits having acute lead poisoning.

When lead is added to whole blood, the intensity of its effect on the red blood cells is markedly less than on washed cells. This is probably explained by the reaction of the lead with the serum and cells simultaneously.

In vitro, cells which have been treated with lead are more fragile than the normal cells, slight trauma causing them to hemolyze.

LEAD STUDIES. III. THE EFFECTS OF LEAD ON RED BLOOD CELLS. PART 2. SURFACE PHENOMENA AND THEIR PHYSIOLOGICAL EXPLANATION. JOSEPH C. AUB, PAUL REZNIKOFF and DOROTHEA E. SMITH, J. Exper. Med. **40**:173, 1924.

Exposure of five billion red cells to 0.01 to 0.05 mg. of lead causes such variations from the normal as: partial loss of normal stickiness, failure of the agglutination reaction when mixed with serum of a different isoagglutinating group, decrease in volume, and loss of normal elasticity.

LEAD STUDIES. III. THE EFFECTS OF LEAD ON RED BLOOD CELLS. PART 3. A CHEMICAL EXPLANATION OF THE REACTION OF LEAD WITH RED BLOOD CELLS. JOSEPH C. AUB and PAUL REZNIKOFF, J. Exper. Med. **40**:189, 1924.

The possible reaction of lead with each of the pure chemical constituents of red blood cells was studied, using as the indicator of chemical combination the effect of lead on the behavior of red blood cells in hypotonic salt solution.

Lecithin, cholesterol, euglobulin, pseudoglobulin, hemoglobin and albumin do not interfere with the effect of lead in increasing the resistance of red cells to hypotonic salt solution. Sodium bicarbonate can neutralize lead but ten times as much is required as occurs in an equivalent neutralizing quantity of serum, therefore, it can play only a small rôle in the reaction.

Inorganic phosphate in concentration normally present in serum neutralizes the same quantity of lead as does the whole serum itself. This can, therefore, completely account for the action of lead on red blood cells.

Adding inorganic phosphate to red blood cells or to whole blood greatly decreases the action of the lead on the cells but cannot eliminate it completely.

When a collodion membrane separates solutions of lead chlorid and phosphate or serum, lead phosphate is precipitated in the membrane with a local formation of acid.

From the data of the three papers, Aub and Reznikoff conclude that a precipitation of insoluble lead phosphate and the formation of acid caused the "leaded" red blood cell to change from an elastic distensible sac to one which is contracted, relatively inelastic and brittle. In such condition the cell can withstand poorly the trauma involved in circulation of the blood, which probably explains the marked destruction of peripheral blood in lead poisoning.

STUDIES ON THE BIOLOGY OF STREPTOCOCCUS. III. AGGLUTINATION AND ABSORPTION OF AGGLUTININ WITH STREPTOCOCCUS SCARLATINAE. FRANKLIN A. STEVENS and A. R. DOCHEZ, J. Exper. Med. **40**:253, 1924.

The authors' conclusions are: Strains of *Streptococcus hemolyticus* from cases of scarlet fever occurring in New York, San Francisco, Chicago, Baltimore and Copenhagen (Denmark) all interagglutinate with immune serums prepared with these strains.

Serums prepared with these strains do not agglutinate pyogenic streptococci or strains isolated from cases of septic sore throat.

The strains obtained from the throats of patients from an epidemic of scarlet fever and the strain from the milk responsible for this epidemic fall into the scarlatinal group according to these agglutination tests.

Absorption tests can be carried out with these strains and serums under proper conditions.

A group of hemolytic streptococci biologically distinct from streptococci from other sources than scarlet fever is constantly associated with scarlet fever. They constitute a group of closely related streptococci which may be identified by agglutination tests.

McCLURE, Evanston, Ill.

A COMPARISON OF THE RATE OF GLYCOLYSIS IN DIFFERENT BLOODS WITH SPECIAL REFERENCE TO DIABETIC BLOOD. F. A. CAJORI and C. Y. CROUTER, J. Biol. Chem. **60**:765, 1924.

The authors found that glycolysis in the blood varies directly with the amount of sugar present; diabetic blood shows no diminished glycolysis, although potassium oxalate tended to decrease it.

SOME EFFECTS OF INSULIN ON THE CARBOHYDRATE AND PHOSPHOROUS METABOLISM OF NORMAL INDIVIDUALS. N. R. BLATHERWICK, MARION BELL and ELSIE HILL, J. Biol Chem. **61**:241, 1924.

The sugar excretion in the urine of normal persons was unchanged by the injection of insulin. Insulin caused a diminution in the inorganic phosphorus content of the blood serum and the inorganic phosphorus excretion in the urine. The authors suggest that this diminution is due to the utilization of the phosphorus in the cells in the metabolism of carbohydrates.

HIGGINS, Cincinnati.

LEAD STUDIES. IX. THE SOLUBILITY OF VARIOUS LEAD COMPOUNDS IN BLOOD SERUM. LAWRENCE T. FAIRHALL, J. Biol. Chem. **60**:481, 1924.

The author compares the solubility of lead, lead oxid, lead carbonate, lead chromate and lead sulphate in water and in horse serum at 25° C. Lead oxid is 100 times more soluble in serum than in water, whereas the other salts show approximately the same solubility as in water. Metallic lead shows some increased solubility in serum. This may explain the reason lead oxid or metallic lead are the common causes of lead poisoning.

LEAD STUDIES. XI. A RAPID METHOD OF ANALYZING URINE FOR LEAD. LAWRENCE T. FAIRHALL, J. Biol. Chem. **60**:485, 1924.

In analyzing the urine for lead, the author adds ammonium hydroxid to the urine; the lead is precipitated with the alkaline phosphates; the precipitate is then collected and ashed, and the lead is determined volumetrically.

HIGGINS, Cincinnati.

PULMONARY DYSPNEA AND ITS EFFECT ON THE PROTEIN CONTENT AND VISCOSITY OF BLOOD SERUM. LUDWIG PETSCHACHER, Wien. Arch. f. Inn. Med. **8**:369, 1924.

The author was able to show that the blood of animals made dyspneic by inhalation of carbon dioxid or excessive pressure from pneumothorax would at a certain stage of the interference with normal gas metabolism show increased viscosity. In spite of this increased viscosity, the albumin-globulin ratio would remain normal; likewise, the total protein concentration. Clinical findings would indicate a similar change in viscosity in human beings suffering from pulmonary dyspnea.

Changes in osmotic pressure and dislocation of ions in the blood are probably responsible for the alterations of the viscosity of the serum. Hydration of protein substance with resulting change in the ionization of the protein bodies will in turn mean alteration in the viscosity. It is possible that disturbances in the gas metabolism can influence the protein of blood serum directly.

THE EFFECT OF INSULIN ON THE BLOOD SUGAR IN MAN. C. ST. RADOSLAV, Wien. Arch. f. Inn. Med. **8**:395, 1924.

The author suggests a diagnostic procedure by which it is possible to differentiate insulin refractory cases from those which react promptly and favorably to the therapy.

The fasting blood sugar of the normal person depends on the carbohydrate and protein content of the preceding food intake. The fat content has no bearing.

In nondiabetic persons, the effect of insulin depends somewhat on the initial value of the blood sugar. In the diabetic patients it is generally proportional to the initial value.

SCHLUTZ, Minneapolis.

OCCURRENCE OF LEPTOSPIRA ICTEROHEMORRHAGIAE IN WILD RATS OF BALTIMORE. GEORGE H. ROBINSON, Am. J. Hygiene **4**:327 (July) 1924.

The incidence of infection of rats with leptospira icterohemorrhagiae ranges from 40 per cent. in localities where human cases occur to 7 per cent. in other localities, with an average infection of about 10 per cent.

The author reports the results of examination of approximately 100 rats from Baltimore. Emulsions of kidney and liver were examined under the dark field

microscope and injected intraperitoneally into guinea-pigs. Leptospiras were found in 7 per cent. of the specimens microscopically, but only four strains produced typical symptoms in guinea-pigs. In no case, microscopically negative, did symptoms develop in a guinea-pig with kidney emulsion.

PRICE, Pittsburgh.

Nutrition

DISTURBANCES OF BONE GROWTH IN CHILDHOOD AND YOUTH. PAUL CAAN, Med. Klin. **20**:886, 1924.

Caan reviews the bone diseases of infants and children in the light of modern roentgen ray and histologic methods. He begins with Calve-Legg-Perthe's disease. An excellent differential diagnosis is presented between osteochondritis deformans, juvenile coxal and tuberculous hip joint disease. The indirect etiologic agent of Perthe's is not known. Some noxa incites a process of bone softening and the pressure produces the deformity. Trauma plays a definite rôle in the destruction of certain cell groups, either by embolism or other interference with the blood supply, or by some other method. Tuberculosis and syphilis may be ruled out of the etiology. Congenital anomalies, inconstant epiphyses, double ossification centers, nuclear hypoplasias are usually considered in the causation. The symptomatology of Schlatter-Osgood's disease is reviewed. It is difficult to distinguish sharply between epiphysitis tibiae and isolated fracture of the tibial tubercle. Trauma is not to be considered as an etiologic factor in the separation of the tibia. The process is rather a local dystrophy within the bone, a growth or calcification disturbance, possibly rachitic or post-rachitic. It may be a sequel of an embolic, a nutritional, an inflammatory process, or the result of a congenital or developmental anomaly.

Köhler's disease (of the os naviculare pedis) may be due to a traumatic or infectious process or both. Köhler also described bone diseases in the ossification center of the patella, in the metacarpal bones (especially cuneiform). Köhler and also Fromme described a lesion of the second metatarsus which Köhler considered to be a disease of the diaphysis. The bone specimen which Fromme described presented callous like tissue, consisting of fibrillary, connective tissue, osteoid tissue, and hyaline bodies. Calve's, Viullet's, Kappe's, Preiser's, Kienback's, and Lehring's entities are mentioned.

EXTRARENAL, ESPECIALLY PULMONARY, WATER EXCRETION. A. F. HECHT, Ztschr. f. Kinderh. **38**:192, 1924.

The water excretion from the lungs can quite easily be measured by the apparatus described by Galcotti. The expired air is passed through weighed calcium chlorid. The apparatus is described. The food concentration (water content) appears to influence the amount of water excreted, except large amounts of liquid taken at one time. Forced breathing, taking of food and physical exercise increase the pulmonary water excretion. There is a definite relation between the body temperature and the amount of water excreted by the lungs. Extensive lung disease and pulmonary congestion decrease the pulmonary water excretion. A combination of mercury and sugar increases, urea hinders the excretion, while atropin has no effect.

PETERMAN, Rochester, Minn.

THE LOWERING OF STOOL ACIDITY AND THE TREATMENT OF CHRONIC DIARRHEA IN CHILDREN BY ADSORBENTS (CHARCOAL, ETC.). ARVO YLPPÖ, Ztschr. f. Kinderh. **38**:313, 1924.

Many adsorbents, such as argilla, animal charcoal, calcium phosphate (tribasic), calcium carbonate, magnesium carbonate, reduce the acidity of

children's stools in a high percentage of cases. The capacity for acid adsorption is increased when several adsorbents are combined. The most active preparation consisted of 8 parts argilla and 2 parts tribasic calcium phosphate mixed with 9 parts argilla and 1 part animal charcoal. This preparation was used in conjunction with protein milk therapy.

DYNAMIC PROTEIN HYPERTHERMIA. H. ZOEPFFEL and W. SCHMITT, Ztschr. f. Kinderh. **38**:327, 1924.

Observations on infants and adults lead the authors to believe that the high temperature following high protein feeding is the result of a physical process dependent on the rapid, complete burning of the protein, or the N containing fraction of the protein. There is a close relationship to the thirst fever. In high fevers of infancy an abundance of protein should be supplied.

THE EFFECT OF HIGH ORAL PROTEIN QUANTITIES ON THE BODY. ILSE REUTER and WALTHER SCHMITT, Ztschr. f. Kinderh. **38**:339, 1924.

Following Zoepff and Schmitt's work (ibid., p. 327), large amounts of protein were fed to adults and infants. The absorption of the protein was good, the stool contained no appreciable amount of N residue. The N balance of the adults, after eleven days with two days of high fever, remained the same. With long continued protein addition there was a definite drop in the hemoglobin and red cell percentages. The refraction index remained about the same. The day after protein feeding the values of the hemoglobin and red cells returned to normal. Thus the results might be interpreted as the effect of hydremia of the blood. A single high protein feeding in infants produced the same results in the hemoglobin, red cell, and serum protein, as did feeding over long periods. With high protein diets the residual blood N was elevated. This rise affected only the urea, the amino-acid fraction remained the same, as did the sodium chlorid, blood sugar, leukocyte and differential counts.

PETERMAN, Rochester, Minn.

CONCERNING INTOXICATION IN NURSLINGS. S. ROSENBAUM, Monatschr. f. Kinderh. **28**:289, 1924.

The literature on alimentary intoxication is full of suggestions as to the loss of body water as an etiologic factor. The writings of Finkelstein, Marriot, Stolte and Kleinschmidt are full of this idea. The author now reports the results of his own studies.

1. His first observations concern the changes in the blood.

(*a*) As regards the water content of the blood, he found in carefully controlled experiments, comparing the blood of the same child when sick and well, and also comparing the blood with that of a well child of the same age, that in all these cases there was a marked diminution of blood water. In some cases of dyspepsia, however, there was a loss of blood water without any of the toxic symptoms. So he concludes that all cases of intoxication have diminished blood water content.

(*b*) The content of the blood in serum albumin would also be a check on the blood water content. In five cases there was almost constantly an increase in the serum albumin. In one or two cases of dyspepsia with increased serum albumin, toxic symptoms did not appear.

(*c*) Examination of erythrocytes in twenty-one observations on eleven nurslings showed in general an increase of 30 per cent. in the count and suggested a marked decrease of blood water.

(*d*) The same holds true for the study of serum viscosity. The cases of intoxication all showed a marked increase. On the other hand, an increase was found in some nurslings with no toxic symptoms, fed on concentrated food.

To summarize his blood work, the author believes there is essentially a loss of blood water which shows itself in increased values of the dry substance, serum albumin, erythrocytes and serum viscosity. In individual cases, however, nurslings show the identical findings without any symptoms of intoxication. Therefore the symptoms cannot be, as Marriot believes, a direct result of blood thickening. Clinical observation and careful recording of the history suggests that the symptoms depend more on the rapidity of the loss of water. Cases in which the loss is gradual do not show such symptomatology.

2. A further study consists of pathologic examination of the water content of different organs.

(*a*) Kidneys. Nothing abnormal was found.

(*b*) Skin. Not enough untreated cases to decide. However, the skin of treated cases shows tendency toward edema.

(*c*) Brain. In three cases there was a diminution in water, in three cases an increase. The latter had been treated with abundant water. The brain is probably like the skin and in these cases shows tendency to edema.

(*d*) Liver. Finkelstein has already described the liver as being the organ in which the injury by water loss brings out the toxic symptoms. Fatty changes were found in the liver in proportion to the water lost. To relate intoxication to fat changes in the liver is not absolutely established, however, as some patients with fatty livers showed no toxic symptomatology.

3. These experiments suggest that giving of water is the main point of the therapy.

(*a*) Giving of fluid intravenously was indicated only for purposes of stimulating the circulation.

(*b*) The intraperitoneal method he has not found so successful.

(*c*) Even the subcutaneous infusion does not do away with toxic symptoms after they have once become established.

In summing up, the writer believes that the best way of giving water is by mouth. When vomiting interferes, water will be better retained if given in very small doses. A great help to this therapy is the suggestion of Bessau to give intramuscular injections of phenobarbital sodium in doses of 0.05 to 0.1 gm. The latter dose is almost always effective and lasts for four hours. The amount of fluid is from 30 to 50 gm. every hour. The best fluid to be given is tea or a 10 per cent. rice gruel.

In spite of this treatment a large number of children died. The death in these cases came after the children were relieved of toxic symptoms and seemed due to heart failure.

In reviewing this article one cannot help feeling that the writer has started with an idea already advocated by others and has attempted to prove his points experimentally. In using his results, however, he uses those which fit his case and discards the others. The results of his therapy showing a loss of 66 per cent. of his cases would suggest that perhaps after all the entire problem has not as yet been solved.

GERSTLEY, Chicago.

ON INTESTINAL INFANTILISM (HERTER). W. PIPPING, Acta Pediat. **3**:342 (June) 1924.

Six cases of this condition are reported. The author's experience with the course of treatment of this condition does not differ greatly from that outlined in most American textbooks. He believes that constitutional factors play a much more important part in the etiology than do exogenous factors. The nervous symptoms, edema, etc., which may accompany this condition are not the result of disturbed nutrition, but are a further manifestation of constitutional inferiority. The deranged nutrition is probably responsible for the infantilism, however.

HOLT, Baltimore.

Prenatal Conditions

ETIOLOGY OF CERTAIN STRUCTURAL DEFECTS. J. BAGG HALSEY, Am. J. Obstet. & Gynec. **8**:131, 1924.

Specific structural defects may be produced experimentally in animals. These defects vary in location according to the time in embryonic life that they are produced and are, to a certain extent, specific and predictable. This is due to the fact that certain structures develop more rapidly at a given time in embryonic life and if injury is produced, they are never able to catch up with the other structures.

The author thinks these injuries are probably in the form of early prenatal localized blood vascular disturbances. The most common defects are eye and kidney defects, club feet, syndactylism, and polydactylism. Blood vascular extravasations in children born by cesarian section indicate that they do occur in the prenatal period independent of birth injuries.

ALLEN, Chicago.

Diseases of the New-Born

THE SYMPTOMATOLOGY OF BIRTH TRAUMA. J. BERBERICK and A. WIECHERS, Ztschr. f. Kinderh. **38**:59, 1924.

A careful study was made of the clinical findings, reflexes, eye signs, and labyrinth tests of 190 children and the findings checked at necropsy. The material consisted of four groups: 1. Children with a birthweight up to 1,800 gm. (twenty-eight). 2. Children with a birthweight of 1,800 to 2,500 gm. (thirty-two). 3. Children over 2,500 gm. (thirty-three). 4. Older infants (thirty-eight), children and idiots of all ages (fifty-nine). The general symptoms of birth injury are the same in all children of all weights. The special otologic examinations have revealed symptoms of value in diagnosis. After establishing normal standards in the Barany and turning stool tests, it was considered that spontaneous nystagmus was a pathologic phenomenon. The reactions to the turning and to cold and hot water injected into the ear are given in great detail. The spontaneous nystagmus usually diminishes and disappears after the second or third month. Older children with spontaneous nystagmus are usually persons with spasmophilia, convulsions, epilepsy or neuropathy. In three cases a vertical nystagmus was found. This was considered a sign of great value. The hemorrhages found in the petrous portion of the temporal bone in new-born infants (Voss) could explain the nystagmus. A definite pathologic condition must exist when ice cold water does not produce the nystagmus reaction. The majority of children who did not react to the caloric tests were those with spasmophilia, idiots, epileptics or neuropaths. On the basis of the new pathologic studies, the findings of Voss, and the results of the present investigation it has been possible, for the first time, to associate the symptoms of birth injury in the new-born with definite pathologic findings. The clinical diagnoses were confirmed by gross examinations in twenty-one cases. The inability always to localize the lesions clinically demonstrates how little is known about the brain, and explains why some children with the most marked symptoms and signs of brain injury survive and reach adult years.

PETERMAN, Rochester, Minn.

ACIDOSIS OF THE NEW-BORN. THE EFFECT OF THE HEMOGLOBIN, CORPUSCLES, CARBON DIOXID AND SALTS ON THE REACTION OF THE BLOOD. ARVO YLLPÖ, Acta Pediat. **3**:235 (June) 1924.

In order to demonstrate acidosis, Yllpö measures the p_H of the serum electrometrically, after removing the carbon dioxid with a vacuum pump. He claims for this method the advantage that it will reveal an acidosis compensated for

by a low carbon dioxid content, whereas direct p_H measurement would reveal only an uncompensated acidosis. The reaction of normal serum is found by this method to vary between p_H 8.5 and p_H 9.2, averaging about p_H 8.9. No change in reaction could be demonstrated in pregnancy. With specimens from the umbilical cord, however, lower values were consistently found, the average being p_H 8.65 and the extremes p_H 8.4 and p_H 8.95. The author feels justified in concluding that there is a tendency toward acidosis in the new-born. A few observations on infants during the first week of life indicated the rapid disappearance of this acidosis.

Apparently this acidosis of the new-born cannot be connected with the high hemoglobin values found at this time. The reaction of the blood is so near the isoelectric point of hemoglobin (p_H 6.9) that hemoglobin can function as an acid only to an insignificant degree. In confirmation of this, the author finds that additions of hemoglobin to serum have little effect on the reaction. It is more likely that variations in the inorganic salt content will explain this acidosis, for it has been shown that umbilical cord blood contains more salts and has a higher ash content than normal blood.

The author also measured the p_H of corpuscles before and after hemolysis with saponin. The saponin regularly produced a marked shift to the acid side. Since this phenomenon cannot be attributed to the hemoglobin, the author is at a loss to explain it and leaves us with the suggestion that some unknown species of acid radicles are liberated by the saponin.

HOLT, Baltimore.

FURTHER OBSERVATIONS OF INTRAUTERINE HEMORRHAGE IN THE NEW-BORN. SIGNIFICANCE OF YELLOW SPINAL FLUID AND OF JAUNDICE IN THESE CASES. WILLIAM SHARPE AND A. S. MACLAIRE, Am. J. Obstet. & Gynec. **8**:172, 1924.

In 100 consecutive deliveries 10 per cent. presented signs of intracranial trauma. Four cases had bloody, and the remaining six yellow spinal fluid. The author feels that the yellow spinal fluid is most probably due to transudation of blood plasma when red corpuscles are absent and to minute hemorrhages when erythrocytes are present.

The etiology of these hemorrhages he divides into three groups: Trauma, congestion, and blood diseases of the new-born. Trauma, he thinks, is by far the most common cause and that syphilis, maternal toxemias, and early low forceps are relatively unimportant.

The anterior fontanel did not appear to be a reliable index of a mild increase in intracranial pressure and he advises manometric readings in all cases of suspected brain hemorrhage. Lumbar puncture in these cases should not only be employed as a diagnostic, but also as a therapeutic measure, repeating from each twelve to twenty-four hours until clear fluid is obtained.

ALLEN, Chicago.

CONCERNING ICTERUS GRAVIS FAMILIARIS NEONATORUM. IVAR THORLING, Upsala Läkaref. Förh. **28**:1, 1922.

The character of this illness is that it attacks several, sometimes all, the children of one family even when the father and mother are normal and the children are full term. The icterus appears early and increases rapidly in severity and is associated with the appearance of bile in the urine, without clay colored stools. Commonly, grave symptoms appear several days after the onset of the icterus and these vary markedly in severity in every case. There is usually weakness with tendency to collapse combined with meningeal symptoms in the form of tonic, less commonly clonic convulsions. Sometimes there are digestive disturbances with enlargement of the liver and spleen. Sometimes a hemorrhagic diathesis has developed where the icterus has lasted longer, but usually death intervenes from weakness before such symptoms occur,

or after an ordinary course free from fever. Rarely do the children recover.

The necropsies show little except marked icteric discoloration of all the organs without obstruction of the bile tracts. Commonly, there is an enlarged spleen. In some cases, hemorrhages on the serous surfaces are found and a somewhat increased transudate in the serous cavities. In the gastro-intestinal tract, occasionally catarrhal changes are mild or marked and swelling and degeneration of the lymphatic follicles are found sometimes as high up as the stomach. The central nervous system shows edema of the pia mater with dilated lateral ventricles and in a few cases, icterus of the nuclei. The author presents cases of two of six children in a family who had undoubted icterus gravis familiaris neonatorum. Both cases were fatal.

The author considers the illness as an intoxication which may develop even before labor is ended. The first manifestation of the intoxication is the icterus, then follows anemia, meningeal irritation, enlarged spleen, irritation of the kidneys, tendency to collapse with subnormal temperature. But this intoxication has no hemorrhages in spite of the prolonged course, no leukocytic infiltrations, and no other marked evidences of degeneration. Blood findings show an anemia and a leukopenia. The fever free course of sepsis is not unusual in children. The failure of a leukocytosis at this period of life cannot be taken as proof against sepsis.

There is no anatomic evidence for the theory, that the disturbance is a result of an abnormal filterability of the liver, as suggested by Ylppö. The icterus alone can hardly be considered as the cause of intoxication, as children with congenital atresia of the bile ducts live for many months, and die of cachexia only after a definite biliary cirrhosis has developed.

The author considers the condition as an intoxication from an infection, though the organism has not been found. The mother's milk can be ruled out, as the condition occurs in children who have never had mother's milk. The presence of icterus at birth on one of his patients suggests the birth canal as a possible source. The fact that the mother of these children is usually healthy precludes the possibility of placental infection and suggests the possibility that the organism which is pathogenic for the child, is saprophytic for the mother and is most probably located in the vagina of the mother. That several children may be born healthy may be due to the fact that the mother has not yet become a carrier of the disease. The portal of entry is most probably the mouth, as the skin, lungs and genito-urinary system show no evidence of infection. We know that at birth the mouth of the child may contain bacteria from the vagina of the mother. The mouth and upper part of the digestive tract show no anatomic changes in icterus gravis neonatorum, but many cases as well as the sixth child in his cases, show enlargement of the lymph follicles of the intestine often as high up as the stomach and we know that such infections occur through the mouth by swallowing infected fluids without causing any local changes in the mouth. Of course not all cases can be explained in this way. Failure of marked degeneration of the organs does not rule out a bacterial infection.

EHRMANN, Chicago.

Acute Infections

ENCEPHALITIS AS A COMPLICATION OF MEASLES. S. HORWITT, Arch. Pediat. **41**:476, 1924.

The author reports a very interesting case of encephalitis occurring on the fourth day of an average case of measles in a boy, aged 6½ years. The onset was sudden, with headache, drowsiness and anorexia ushering in the disease. Convulsions were followed by coma. The reflexes showed a tâche cerebrale, sluggish pupils, absent abdominal and knee jerks, Babinski and

Gordon signs on the right, rigidity of the neck with Kernig and Brudzinski signs. Choked disc was marked. Spinal puncture showed pleocytosis with 95 per cent. mononuclears, albumin one plus, globulin one plus, culture and smear negative for organisms. During the course of the disease there were sudden lucid intervals succeeded quickly by profound stupor. Return to normal was gradual and ultimate recovery was complete.

PERITONITIS IN SCARLET FEVER. E. S. PLATOU, Arch. Pediat. **41**:535, 1924.

The author reports a very interesting case of a girl, aged 3 years, who on the tenth day of scarlet fever developed a profuse vaginal discharge coincident with a sharp rise in temperature. Smear showed a gram-positive diplococcus. Otitis media developed and on paracentesis the temperature dropped, but never quite to normal. There occurred a sharp rise again to 105 on the twenty-fifth day associated with abdominal pain and dysuria. Urine showed albumin and pus. There was tenderness above the bladder region. Finally a mass was felt on rectal examination. An operation through the rectum drained a small amount of pus. The child developed pneumonia and died on the tenth day after operation. At necropsy subphrenic and pelvic abscesses were found.

SUGGESTING THYMIC INVOLVEMENT IN PERTUSSIS. W. N. ADKINS and J. W. LANDHAM, Arch. Pediat. **41**:580, 1924.

It is pointed out that there are a great many features which pertussis and status lymphaticus have in common, viz., the spasmodic manifestations, asthenia, breath-holding spells, congestion and cyanosis of the face, absence of febrile reaction, high lymphocyte count in the blood and finally the fact that roentgen-ray treatment is of benefit in both conditions. The authors suggest that roentgen-ray studies be made of the thymus in pertussis cases before and after roentgen-ray therapy and that the therapy be made by exposures over the thymus region. Originality is claimed for these thoughts and suggestions.

VON MEYSENBUG, New Orleans.

CONCERNING ANOXEMIA IN PNEUMONIA, ESPECIALLY THE CROUPOUS TYPE. CHRISTEN LUNDSGAARD, Ugesk. f. Laeger **30**:561, 1924.

The author points out that in a considerable number of patients with pneumonia there results a deficient oxygenation of the arterial blood and consequently a decrease in the oxygen content and tension of the capillary blood. If this oxygen deficiency becomes extreme, a series of symptoms arises which is known as anoxemia.

The most apparent symptoms are cyanosis, an increase of the act of respiration, and a slowing of the action of the heart muscle due to abnormal stimulation of the nerve centers.

With the aid of a properly conducted therapy of oxygenation, the symptoms caused by anoxemia may be diminished or made to disappear.

SCHLUTZ, Minneapolis.

PIRQUET'S MEASLES THEORY IN LIGHT OF AN INFLEXIBLE BIOLOGIC LAW OF FRACTIONED SATURATION. E. MAYERHOFER, Ztschr. f. Kinderh. **38**:244, 1924.

Pirquet's measles theory is quoted and compared with the budding and growing of trees. The measles reaction theory is considered a phenomenon of a universal, inflexible biologic law which can be observed in every lymph stream of every animal and in the circulatory system of plants.

SCARLET FEVER AND THE VEGETATIVE NERVOUS SYSTEM. WALTER BLOCK and ERNST KOENIGSBERGER, Ztschr. f. Kinderh. **38**:350, 1924.

All of the children with scarlatina studied showed a hyperexcitability of the parasympathetic nervous system, which raises the question whether "vagotonias" are predisposed to scarlatina or whether vagotonia is a sequence of scarlatina. The chronic hyperthemias of scarlatina convalescents are especially conditioned by this hyperexcitability, since they may be reestablished by epinephrin given intravenously.

PETERMAN, Rochester, Minn.

THE PREVENTION OF SCARLET FEVER. GEORGE F. DICK and GLADYS HENRY DICK, J. A. M. A. **83**:84, 1924.

This article gives a rather interesting review of the work done by the Dicks in which they found a streptococcus that fulfilled all the requirements of Koch's laws. The discovery of a toxin offered a scientific basis for: (1) the development of a skin test for susceptibility to scarlet fever; (2) preventive immunization and (3) the production of an antitoxin.

THE USE OF GENTIAN VIOLET IN DIPHTHERIA CARRIERS. J. SHELLEY SAURMAN, J. A. M. A. **83**:249, 1924.

Five cases are reported in which throat cultures became negative for Klebs-Loeffler bacilli, soon after the following treatment. The patient flushes the nares and fauces with a light saline solution on awakening, after each meal and once during the evening. Immediately after this, the nares and fauces are well sprayed with a 2 per cent. aqueous solution of gentian violet in the ordinary atomizer.

Cultures are taken immediately preceding one of the treatments, and as a check, no treatment is given for forty-eight hours following the second negative culture. The culture is then repeated.

Although the dye stains everything it comes in contact with, it will leave no permanent discoloration of the tissues.

THE DICK TEST IN NORMAL PERSONS AND IN ACUTE AND CONVALESCENT CASES OF SCARLET FEVER. IMMUNITY RESULTS WITH SCARLET FEVER TOXIN. ABRAHAM ZINGHER, J. A. M. A. **83**:432, 1924.

The Dick test is a reliable index of immunity and susceptibility to scarlet fever. In conjunction with active immunization with scarlet fever toxin, it will help in solving the problem of the control of scarlet fever. The test is valuable in that within twelve hours time, there may be detected those susceptible persons who need immediate passive immunization with scarlet fever antitoxin.

The test is of value in the diagnosis of doubtful cases of scarlet fever. Where the reaction is strongly positive early in the disease and again in convalescence, it speaks against the diagnosis of scarlet fever. A definite negative reaction in the first two days of the disease should place one on guard against diagnosis of scarlet fever. Other applications of the skin test consist in studying the specific character of the toxin produced by different strains of hemolytic streptococcus.

A new impulse will be given by the application of the test to the study of scarlet fever to determine whether the disease includes a single entity or whether, as was long suspected, the clinical conception of scarlet fever includes a variety of diseases with similar symptoms, but not necessarily associated with the presence of the specific strains of the hemolytic streptococcus of scarlet fever.

Scarlet fever is a combined toxemia and bacterial infection. The immunity is antitoxic and not to any extent antibacterial. This is shown by the fact that convalescent patients who give a negative Dick reaction, may develop secondary septic complications produced by the specific streptococcus.

The results with the Dick test in normal persons indicates that the percentage of susceptible persons by age groups and by social status resembles closely the similar percentage of persons susceptible to diphtheria noted with the Schick test.

Of 170 scarlet fever patients tested during convalescence, 158, or 93 per cent., gave a negative Dick reaction. There was some doubt about the diagnosis of scarlet fever in most of the twelve patients.

Active immunization with scarlet fever toxin is a safe procedure, and is not to any extent associated with the development of constitutional symptoms.

Purification of the toxin gives a preparation that is better for purposes of the test and active immunization, as it eliminates the pseudo element of the Dick test. The scarlet fever toxin is neutralized in multiple proportions by the antitoxic serum.

HABITUAL HYPERTHERMIA DURING RECOVERY FROM SCARLET FEVER. HEINRICH FINKELSTEIN, J. A. M. A. **83**:611, 1924.

Finkelstein believes that the elevated temperature that begins in the third or fourth week of the disease, and which can not be explained by any possible physical finding, is perhaps due to a balance disturbance in the autonomic nervous system or in the heat regulating center.

The experience that, in a few cases, a single injection of epinephrin removed the hyperthermia permits of the hope that an effective method of treatment will be found.

HENN, Chicago.

Chronic Infections

THE EVOLUTION OF JUVENILE TUBERCULOSIS. HENRY D. CHADWICK, Boston M. & S. J. **191**:252 (Aug. 7) 1924.

The author gives such a clear yet concise elucidation of the origin of tuberculosis in childhood, and of the processes of its extension that one should read the original article if possible. He again emphasizes the fact that fatigue is the most common early symptom, especially in those underweight or failing to gain normally; that the cough is a later symptom, usually due at first to the pressure of the glandular masses on the bronchi rather than to bronchitis, and therefore unproductive; and that the positive D'Espine sign, roentgen-ray confirmation and positive intracutaneous tuberculin test should be looked for as corroborative evidence.

GENGENBACH, Denver.

THE DIAGNOSIS OF ACUTE MILIARY TUBERCULOSIS IN INFANCY. HERBERT KOCH, Ztschr. f. Kinderh. **38**:232, 1924.

In two cases the diagnosis of miliary tuberculosis was made on the symptom complex of cyanosis, dyspnea with negative findings in the heart and lungs, but with enlargement of spleen and liver and a high fever. The tuberculin and the roentgen-ray tests were negative.

THE CUTANEOUS TUBERCULIN TEST IN MENINGITIS TUBERCULOSA AND GENERAL MILIARY TUBERCULOSIS. PAUL OESTREICHER, Ztschr. f. Kinderh. **38**:359, 1924.

Three hundred twenty-five case histories of tuberculous meningitis and general miliary tuberculosis were reviewed. Ninety-two histories were of value for statistical study; of these, eighty-two were of tuberculous meningitis

and nine of miliary tuberculosis. About one-third of the cases of tuberculous meningitis and miliary tuberculosis gave a positive tuberculin reaction; one-third gave a negative reaction. The reactive ability of the body toward tuberculin is markedly diminished in the third and fourth weeks of the disease. Cachexia does not seem to be a factor in the reaction. In the first year of life the reactions were all positive, later the age had no influence (in children). There was no difference between the sexes. Cases with more cerebral symptoms were less often found to have a tuberculin reaction. The fever reaction and the tuberculin reaction are not parallels. The highest number of positive reactions were found in the coldest seasons. The tuberculin reaction is of no prognostic value. Previous infections do not influence the reaction. The diazo reaction was negative in 86 per cent. of the cases where there was positive tuberculin reaction.

PETERMAN, Rochester, Minn.

SUGGESTIONS FOR THE TREATMENT OF PULMONARY TUBERCULOSIS BY THE ULTRAVIOLET RAY. W. B. CHAPMAN, J. Radiol. **5**:237, 1924.

Expose to a deep therapy lamp for ten minutes, front and back. This causes free perspiration, increases the peripheral circulation, and places the skin in a more receptive condition. Following a vigorous rub to dry the skin and still further increase the circulation, the patient is exposed to the ultraviolet rays. As blonds have less tolerance for the ray they are exposed for one minute only, but brunettes are given two-minute initial exposures. The quartz tube is set 30 inches from the patient's body. The time is increased one minute at each exposure until ten-minute exposures are given. The distance from the tube to the patient is then diminished from 1 to 2 inches at each exposure until it is 15 inches from the patient. Exposures should be given daily when possible, except in the presence of a marked skin reaction, when they should be discontinued until the erythema subsides. Exposures given three times weekly are quite satisfactory. In the author's experience from one to three months are necessary to arrest a case of pulmonary tuberculosis, depending on the severity of the case. Increased expectoration and coughing may be observed shortly after beginning the treatment. Stress is placed on the detailed care of the patient while under treatment. Diet, rest, exercise, and general hygiene, together with physical examinations and indicated medicinal measures, must not be neglected if the most satisfactory progress is to be made.

Three case reports of three patients are presented in some detail, all of whom had tubercle bacilli in the sputum. In addition, one had a tuberculous hip. All made very satisfactory progress. In conclusion, the author states that the ultraviolet ray, while not specific, is the most valuable recent contribution to the treatment of pulmonary tuberculosis.

CHAMBERLAIN, Chicago.

TUBERCULOSIS IN INFANTS. A. B. MARFAN, Le Nourrisson **12**:153 (May) 1924.

Tuberculosis in infants is always an acquired disease, never congenital. The most frequent source of infection is the sputum from an open case of the disease. This is especially true when the mother or nurse is infected. The bacilli are carried in the droplets produced in talking, sneezing, kissing and coughing; these droplets often travelling a distance of a meter. One other important source of contagion is the milk from tuberculous cows. Formerly such milk was responsible for 8 per cent. of the cases occurring in infants under 2 years, but today under improved conditions of sanitation, handling and pasteurization, scarcely 2 per cent. of cases can be traced to this source. Boiling milk for 2 or 3 minutes suffices to destroy the tubercle bacilli.

The common portal of entry is through the respiratory tract, but may be through the digestive tract or through the skin. When the infection takes place through inhalation, the original focus is usually found in the right middle lobe, or in the upper part of the lower lobe and not at the apex of upper lobe, as occurs so commonly in adults. From the lung focus, the bacilli are carried through the lymphatics to the tracheobronchial glands, which are often greatly enlarged. Sometimes the infection gains entrance through the upper respiratory passages, especially when some pathologic condition is present such as ulcerations around erupting teeth, or diseased tonsils and adenoids. In such cases the bacilli proceed to the submaxillary and cervical glands, producing the tuberculous glands of the neck seen so often in cases of scrofula. It may even be possible for the tuberculous infection to enter through the conjunctiva. Experimental infection in guinea-pigs can be initiated in this manner, although the eye itself shows no involvement.

When the infection enters through the digestive tract, the mucosa may remain intact, the first manifestations appearing in the mesenteric glands.

Infection by way of the skin is possible in cases of eczema, impetigo or through some superficial abrasion. Cases have been reported following ritualistic circumcision, or after piercing the ears for earrings.

In pulmonary cases, except in cases of massive infection, the initial lesion is often small and insignificant, the principal involvement being found in the tracheobronchial glands, which become greatly hypertrophied. The outcome in these cases varies. Spontaneous cure may occur or there may be extension to adjacent structures with signs of compression, or caseation may ensue with rupture into a blood vessel, giving rise to miliary tuberculosis. Suggestive symptoms of tracheobronchial gland involvement are slight fever (from 37.5 to 38.5 C.), mild bronchitis, loss in weight, anemia and digestive disturbance. Add to these a positive tuberculin skin test and the diagnosis is established. Roentgen-ray examination is helpful, but more particularly from the point of view of prognosis. If the adenitis gradually subsides an active immunity is started which affords protection in adult life. Such a cure is rare in infants under 1 year. More frequently at this age the infection becomes generalized, the final picture being miliary tuberculosis, with tuberculous meningitis.

McCLELLAND, Cleveland.

PROGNOSIS OF TUBERCULOSIS IN INFANCY. M. S. REUBEN and ALONZO D. SMITH, Arch. Pediat. **41**:529, 1924.

The prevailing opinion at present is that the prognosis of tuberculosis in infancy is not as grave as it was formerly thought. There are two facts with regard to tuberculosis in infancy which are undeniable: 1. The younger the infant the worse the prognosis. 2. In infants, as a rule, the process is localized in the lungs and the bronchial glands and there is a great tendency for the process to become disseminated into a general miliary tuberculosis or tuberculous meningitis; but the authors contend that even in infants under certain circumstances the process tends to become localized and arrested. The four main factors on which the severity and course of the infection depend are: the dosage of infection and resistance of the body; the weight of the child; the nature of the feeding, whether breast or bottle fed; the hygienic surroundings of the child, favorable or not.

Roentgen-ray pictures showing miliary tuberculosis of the lungs of an infant are given at successive intervals covering a year, and the gradual healing of the process is indicated although the infant finally succumbed to tuberculous meningitis.

VON MEYSENBUG, New Orleans.

Internal Diseases

EXPERIENCES WITH INSULIN TREATMENT IN CHILDHOOD. RICHARD PRIESEL and RICHARD WAGNER, Ztschr. f. Kinderh. **38**:103, 1924.

Severe diabetics must undergo protein restriction even with insulin. Blood sugar determinations are necessary in childhood. In diabetic coma large doses of insulin and glucose and liquids are indicated. The danger of overdosage with insulin is greater in childhood than in adulthood. The carbohydrate tolerance of the person may be raised by insulin treatment if the disease has not progressed too far. The intoxication following overdosage with insulin is quite similar to the picture produced in Rowntree's "water intoxication."

THE RELATION BETWEEN THE IMPULSE TO URINATE AND THE BLADDER CONTRACTION IN ENURESIS IN CHILDREN. SAMUEL AMBERG, Ztschr. f. Kinderh. **38**:169, 1924.

Whether enuresis is considered a psychic disturbance or not, it involves an abnormal emptying of the bladder. It is necessary to determine whether there is a difference between micturition and its mechanism in enuretic children and in normal healthy children. The apparatus for emptying and filling the bladder and measuring the bladder pressure is described. An electric switch enables the child to register when the bladder feels full and when the desire to urinate is felt. Twenty-five observations were made on eleven boys and seven girls. There were few enuretic children who had no urinary desire or contraction of the bladder. Every contraction of the bladder is not accompanied by a desire for micturition. The desire may accompany the contraction. This occurs in the majority of cases. There may be a latent period between the desire and the contraction or vice versa. We do not have enough information to establish a theory of micturition. In most of the enuretic patients there was no desire to urinate during the bladder contractions. At least there is a latent period between the contraction and the desire.

PETERMAN, Rochester, Minn.

ROENTGENOLOGIC DIAGNOSIS IN DISEASES OF THE ALIMENTARY TRACT. N. J. NESSA, J. Radiol. **5**:232, 1924.

Report is made on the relative frequency of gastro-intestinal conditions as observed by twelve gastro-enterologists on a total of about 30,000 cases. Pathology in the alimentary tract was diagnosed in about 64 per cent. of these cases, being divided as follows: appendix, 30 per cent.; gallbladder, 15 per cent.; duodenum, 15 per cent.; stomach, 10 per cent.; colon, 7 per cent.; esophagus, 3 per cent.

Normal roentgen-ray findings with an outline of the points to be observed is given. An outline of abnormal findings is also given, both direct and indirect (direct being filling defects). Gastric ulcer and carcinoma, appendix, gallbladder, and duodenal ulcer findings are outlined in more detail.

In conclusion, the author states that properly obtained and estimated roentgen-ray findings (together with the clinical and laboratory findings) are of great value in gastro-intestinal diagnosis.

CHAMBERLAIN, Chicago.

THE PATHOGENESIS OF ANAPHYLACTIC ASTHMA. KAJ BAAGÖE, Acta Pediat. **3**:369 (June) 1924.

Baagöe considers that the constriction of the bronchioles in asthma is caused by direct action of the antigen on the mucosa, producing hyperemia and edema. The hypothesis that the constriction is due to spasm of the bronchial muscu-

lature is based chiefly on pathologic observation on guinea-pigs whose anaphylactic reactions differ in many respects from those of man. The clinical observations which lead the author to this conclusion are:

1. Simultaneous production of urticaria and asthma by injection of specific antigens. 2. Cases in which the antigen was inhaled. Symptoms appeared first in the upper respiratory tract, passing downward and terminating with typical asthma. Direct observations on the upper respiratory passages revealed edema and hyperemia, which presumably caused the symptoms lower down.

HOLT, Baltimore.

PROGNOSIS OF MYXEDEMA IN CHILDHOOD. E. LAZAR and E. NOBEL, Ztschr. f. Kinderh. **38**:235, 1924.

Six cases of myxedema were studied after several years of treatment. They all showed some physical signs of myxedema. The intelligence of these patients varies quantitatively. The thinking power is retarded. The short, simple, routine reactions are almost normal, but the more difficult situations, the judgment complexes are not well met. The quantitative difference is entirely dependent on the stage when treatment was begun and on the regularity of the treatment. Complete cure with normal development would be possible if the treatment was started early enough (?).

DIATHERMY IN THE TREATMENT OF PYLOROSPASM IN NURSLINGS. W. TOBLER, Ztschr. f. Kinderh. **38**:285, 1924.

Two infants with pylorospasm were given intensive diathermic treatments daily. The results were very good; the regurgitation was stopped and the infants were much quieter.

INFANTILE SCURVY IN VIENNA, 1916-1923. HANS WIMBERGER, Ztschr. f. Kinderh. **38**:293, 1924.

The stringent restrictions during the war brought about a marked increase in infantile scurvy. The peak of the curve fell in May, 1921, so that the quality of the milk rather than the quantity was directly responsible. The poor quality of the milk was due to the scarcity of fodder for the cows. Most of the patients were infants between 8 and 10 months of age.

PETERMAN, Rochester, Minn.

CONGENITAL HYPOGONADISM. M. S. REUBEN and G. R. MANNING, Arch. Pediat. **41**:489, 1924.

In a survey of eight cases of hypogonadism, the following characteristics were noted: the children were oversized and overweight for age; the genitals were under developed, secondary sexual characteristics were absent. The mentality was normal in all. The part below the unbilicus was larger in proportion to total length than in normal children. The thymus in several cases was persistent, the pituitary normal or slightly enlarged. They all presented contours of a feminine type, associated with adiposity. The roentgenogram of wrists showed delayed ossification. The basal metabolism in two cases was subnormal and the sugar tolerance in two cases was normal.

GAUCHER'S DISEASE. MARK S. REUBEN, Arch. Pediat. **41**:456, 1924.

Report of a case of Gaucher's disease in a boy, aged 15 years, with detailed historical sketch and description of the etiology, pathology, symptomatology and course of the disease. Differential points between Gaucher's and splenic anemia, Banti's and von Jaksch's anemia are tabulated.

VON MEYSENBUG, New Orleans.

THE INSULIN TREATMENT OF DIABETES MELLITUS. W. FALTA, F. DEPISCH and F. HÖGLER, Wien. Arch. f. Inn. Med. **8**:13, 1924.

The problem in the diabetic patient is threefold. Get him sugar and acetone free, bring about a return to normal of the blood sugar, and permanently establish a tolerance for carbohydrate. During the accomplishment of this program, the nutrition of the subject must be carefully guarded and maintained.

The authors raise the question whether simultaneous treatment with insulin and dietetic measures will accomplish the objective more quickly and with less hardship to the patient than either method used alone. Is the end effect of the combined treatment better and more permanent and can favorable results still be looked for in cases in which the dietetic treatment has proved insufficient?

The results of their clinical observations seemed to show that with the combined treatment it was possible to attain an adequate carbohydrate tolerance in a shorter time than was possible with dietetic treatment alone. They could bring about this tolerance even with moderate additions of carbohydrate to the food. It was further shown that with the combined treatment, tolerance could be still established in cases where dietetic measures alone had completely failed.

The rapidity with which insulin treatment overcomes acidosis and its superiority in this respect over dietetic treatment alone was again demonstrated in their series of cases.

SCHLUTZ, Minneapolis.

CLINICAL STUDIES OF SOME DISEASES OF THE BLOOD IN CHILDREN, WITH ILLUSTRATIVE CASES. J. P. CROZER GRIFFITH, Arch. Pediat. **41**:511, 1924.

As a summary of the information which may be gathered from details of the cases which the author reports, the following conclusions are drawn:

1. That the blood picture of chlorosis is of very frequent occurrence in infancy and childhood and that such cases should properly be designated as chlorosis.

2. That the administration of iron, without any other treatment, is often of striking benefit in these cases and in simple anemia.

3. That in diseases of the blood in general, certainly in childhood, the symptoms appear to be, to a certain extent, interchangeable, or at least kaleidoscopic.

4. That without an examination of the blood a diagnosis is more likely to be mistaken than correct, and that even with it a diagnosis cannot be based on any one hematologic feature; certainly not on alteration in the character of the red cells; any excess either of lymphocytes or myelocytes, a high color index or a low color index.

5. That there appears to exist no definite relationship between the degree of alteration of the blood and that of the size of the lymphatic glands and the spleen.

6. That variations in the blood picture occur not only between different cases, making the differentiation of the disease present difficult, but from time to time in the same case.

7. That, in fact, only careful and frequent examinations of the blood and of the symptoms in general insure our reaching a correct diagnosis.

VON MEYSENBUG, New Orleans.

THE PATHOGENESIS OF ANAPHYLACTIC ASTHMA. KAJ. BAAGE, Ugesk. f. Laeger **31**:577, 1924.

In the organism subject to anaphylactic asthma, the asthmatic attack is a symptom coordinated with other symptoms of the anaphylactic condition.

The attack, as well as other symptoms, may be ascribed to a peripheral vasomotor disturbance induced by antigens having a hyperemic and edema-forming characteristic.

The asthmatic attack may thus evidently be due only to a purely peripheral vasomotor disturbance.

It is often accompanied by a bronchial spasm. This is probably also due to peripheral influences.

INVESTIGATIONS CONCERNING THE MANNER OF ACTION OF INSULIN, I. CHRISTEN LUNDSGAARD and SVEND AAGE HOLBØLL, Ugesk. f. Laeger **32**:591, 1924.

The formation of γ glucose in a solution of β glucose, to which insulin and a muscle preparation have been added, is demonstrated by the comparison of polariscope readings. The extent of this conversion may reach 76 per cent., but further formation of γ glucose is prevented due to the reversibility of the reaction.

This muscle preparation is most active when fresh and at 37 C. It is inactive after a lapse of two hours and when subjected to a temperature of 20 C.

SCHLUTZ, Minneapolis.

Nervous Diseases

THE PATHOGENESIS OF CEREBRAL DIPLEGIA. JAMES COLLIER, Brain **47**:1, 1924.

Collier reviews at length the literature on the pathogenesis of cerebral diplegia since the publication of Little's classic monograph in 1861. His clinical data, based on a clinical experience of 200 cases, is so complete and free from error of observation or deduction as to stand the tests of time. Little established the fact that cerebral diplegia is often associated with abnormalities of birth and "assumed the affection to be a widely spread fine lesion of the cerebral substance, which could affect one part of the brain more than another." He excluded gross birth injuries as causal factors of the diplegic form of spastic paralysis. Many of his successors have not proved equally successful in their observations and deductions.

The work of Charcot, James Ross, McNutt, Strumpell, Brissaud, and many others are discussed. Freud's monograph of 1897, Collier considers still the most authoritative exposition of the subject. Freud was the first to term a bilateral symmetrical defect of the brain "cerebral diplegia," and to prove that all diplegias dating from birth and attributed to abnormalities of birth really had their pathologic origin in intra-uterine life.

Collier concludes that the essential anatomic cause of diplegia is a primary degeneration of the cerebral neurons from causes at present elusive, except in rare cases of syphilitic infection of brain. He believes this conception of the pathogenesis compatible with our present pathologic evidence and explains best the symptomatology and clinical aspects of this class of diseases.

ELECTROMYOGRAPHIC STUDIES OF EXPERIMENTAL CONVULSIONS. STANLEY COBB, Brain **47**:70, 1924.

Cobb reports the application of string galvanometer to the study of thujone convulsions in rabbits. He believes the experiments important, as clinicians usually describe tonic and clonic periods of an epileptiform convulsion as distinct phenomena. Analogy to human epilepsy should be drawn with reservations, but he considers it reasonable to apply this rabbit physiology to the human being, having both mammalian nerve muscle mechanisms. His electromyograms indicated that voluntary muscular contractions, clonic and tonic convulsive contractions, phasic and postural reflexes are all tetanic in character.

THE SEQUELAE OF ENCEPHALITIS LETHARGICA. A. G. DUNCAN, Brain **47**:76, 1924.

This report represents a very thorough investigation of eighty-three survivors of undoubted encephalitis lethargica in the London Hospital followed for eighteen months after the onset. Sequelae are classified as mental and physical. The author found that children under 10 years were most liable to severe mental sequelae, since arrested and impaired development may be added to actual brain damage. This liability diminished in the next two decades. Duncan found that early and marked negative mental signs (lethargy, drowsiness and coma) in the acute stage likely to be associated with severe mental sequelae while positive mental signs (restlessness, delirium and mania) did not have the same significance.

Severe physical sequelae were less common in children than in adults between 20 and 40. When physical sequelae appeared several months after the illness, the author found no connection between the acute stage and the severity of sequelae. "The severe and occasionally fatal nature of the sequelae, their irregular clinical course with remissions and relapses, strongly suggest that in some cases the virus of encephlitis lethargica continues to exist in the nervous system for long after the acute symptoms have subsided."

PYKNOLEPSY: A FORM OF EPILEPSY OCCURRING IN CHILDREN WITH A GOOD PROGNOSIS (ABRIDGED). W. J. ADIE, Brain **47**:96, 1924.

Adie recommends the name "pyknolepsy" for "a disease with an explosive onset between the ages of 4 and 12 years, of frequent short, very slight, monotonous minor epileptiform seizures of uniform severity, which recur almost daily for weeks, months or years, are uninfluenced by antiepileptic remedies, do not impede normal mental and psychical development, and ultimately cease spontaneously never to return."

He states that nothing has been written on this subject in English, though since Friedmann first described the disease in 1906 it has been frequently discussed in foreign journals, etc. He cites the normal after history of his own and many foreign cases. "Pyknolepsy" should be applied only to cases conforming strictly to his descriptions; deviations would cast doubt on diagnosis. He emphasizes the point that there is a form of epilepsy in children with distinct clinical features and good prognosis.

A FORM OF FAMILIAL CEREBRAL DEGENERATION CHIEFLY AFFECTING THE LENTICULAR NUCLEUS. DONALD PATERSON and E. ARNOLD CARMICHAEL, Brain **47**:207, 1924.

Paterson and Carmichael have presented in a very complete manner what they consider to be a new form of familial cerebral degeneration. Histories and pathologic examinations of two remarkable cases are reported. The family history is of interest. Only two children have survived twelve pregnancies. Death occurred from the third day to the ninth month, with the exception of the sixth child, who died at the age of 2 years. Diagnosis of amaurotic family idiocy was made by various children's specialists on the eight patients whose deaths preceded those of the two reported, though the characteristic cherry red spot in the fundus was apparently not found. The father's history was negative and the Wassermann test on father, mother and two children was entirely negative. The only points in the mother's history worthy of note were a leukorrheal discharge, which appeared before her marriage and persisted even during pregnancy, and a peculiar and unbalanced mental outlook during pregnancy in marked contrast to her sane view of life at other times. Her family history was otherwise negative.

Pathologic findings in the two children were remarkably similar. "The severest pathologic changes are found in the lenticular nuclei, while the cortex

is badly developed and degenerated. Clinically, it gives rise to apathy and apparent blindness without changes in the fundi of the eyes." And "there is an absence of paralysis and abnormal movements."

The authors conclude as a result of their observations "that an infective organismal state at the base of the brain has occurred, with a consequent toxemia which affected chiefly the cells of the cortex." It seems probable that this infection was antenatal and resulted from the chronic endometritis of the mother, which passed by way of the placenta and cord to the fetus, where it set up a basal meningitis. Since the mother refused a gynecologic examination, and microscopic examination of placenta and cord of the younger child was not done, the causal agent and route of infection of this familial cerebral degeneration are unknown.

LUCAS, San Francisco.

OBSERVATIONS IN THE DIAGNOSIS AND TREATMENT OF BRAIN INJURIES. WILLIAM SHARPE, Northwest Med. **23**:308 (July) 1924.

The presence or absence of increased intracranial pressure is of more significance in brain injury than the presence or absence of fracture of the skull. Patients should be allowed to react from shock before prolonged neurologic tests or spinal punctures are performed. Spinal drainage is best, provided the pressure is not greater than twice the normal, in which case operation is usually necessary. Operation should never be done during the initial period of shock or in the terminal period of medullary edema. Patients showing chronicity of symptoms with increased intracranial pressure usually show at necropsy or operation a wet edematous brain, which results because the organization of hemorrhage about the supracortical veins has impaired their capacity of absorbing the excess of spinal fluid. The same findings have been found in cases of cerebral spastic paralysis with mental retardation in children. Operative procedures are less frequently necessary in children because of the rarity of cerebral edema, and a better compensating mechanism of absorption and secretion of the fluid. Operation was performed in 16 per cent. of cases under 12 years and in 30 per cent. over 12 years. Also the prognosis in childhood is much more favorable.

The younger the age at which the acute brain injury occurs, the greater the similarity of the resulting condition to the cerebral spastic paralysis due to intracranial hemorrhage in the new-born. In the younger patients physical and mental retardation are due to the increased intracranial pressure occurring before the higher centers were fully developed. Lowering of pressure by the administration of thyroid and thymus extracts was successful in but 8 per cent. of cases. Subtemporal decompression and drainage caused marked improvement in the majority of these patients. Operation should be performed before the hemorrhagic residue can organize. The ideal time for treatment is during the acute stage of the hemorrhage, when it can often be drained by spinal puncture, before the blood coagulates. Repeated spinal punctures should render the fluid clear and the pressure normal. If it does not, decompression should be done. Clinical signs of hemorrhage in the cranium of the new-born may be nothing more than a slight drowsiness, difficulty in nursing and an occasional slight muscular twitching.

SCOTT, Philadelphia.

THE NEUROSIS QUESTION IN CHILDHOOD. JULIUS ZAPPERT, Ztschr. f. Kinderh. **38**:109, 1924.

Zappert continues his argument with Pototzky on the classification of the neuroses. Neuropathy and psychopathy are the bases on which the other neuroses may develop, but they are not the unconditional forerunners of these neuroses. Some neuroses may develop in persons who have not had nervous or psychopathic manifestations and who do not develop them.

PETERMAN, Rochester, Minn.

THE BISEASONAL PREVALENCE OF INFANTILE PARALYSIS (ACUTE ANTERIOR POLIOMYELITIS). W. J. AYCOCK and PAUL EATON, Am. J. Hygiene **4**:356 (July) 1924.

Aycock and Eaton made a study of the seasonal prevalence of infantile paralysis in the United States over a period of eleven years. In addition to the familiar summer rise in the number of cases, they found, to a less extent, a rise occurring quite regularly during March and April, with May showing the least number of cases during the year. This marked contrast in the seasonal occurrence of infantile paralysis, according to the authors, would indicate the possibility of two modes of transmission of the condition.

No suggestion is made as to what these two modes might be.

PRICE, Pittsburgh.

Diseases of the Eye

CONGENITAL CORNEA PLANA. WILBUR E. SWEET, Am. J. Ophth. **7**:437 (June) 1924.

Sweet reports a case of congenital cornea plana. This is a very rare anomaly and is often confused with microcornea. The boy, aged 7 years, complained of very poor vision. His family history could not be traced, as he was an orphan. The frontal section of both eyes was flat, the cornea vertically oval. There was no distinct limbus, but rather a blending of the sclera into the corneal tissue. There was an increased vascularity at the limbus. The anterior chamber was so shallow that the iris appeared to lie in contact with the posterior surface of the cornea. Complete examination revealed no other anomalies of the eyes.

WESCOTT, Chicago.

Diseases of the Nose and Throat

BACTERIOLOGIC STUDY OF MIDDLE EAR INFECTIONS. EUGENIA VALENTINE, J. Infect. Dis. **35**:178 (Aug.) 1924.

This report concerns itself with an intensive study on the bacteriology of 100 acute middle ear infections in seventy-seven cases, and of twelve cases of chronic middle ear infection.

It was found that the beta hemolytic streptococcus is probably the most important etiologic organism in the majority of acute middle ear infections in San Francisco. In a small percentage of cases, staphylococci, pneumococci, green (alpha) streptococci, and diphtheroid bacilli predominate.

The organisms obtained from twelve chronic cases, in pure or mixed culture, include diphtheroid bacilli, staphylococci, *B. pyocyaneous, B. proteus* and *B. coli.*

A study of the throat flora in relation to the bacteria found in the ear discharges of acute middle ear infections reveals that, in cases in which the beta hemolytic streptococcus, pneumococcus, or green (alpha) streptococcus are the apparent exciting factors in the ear discharge, the same bacterial species are found in the throat.

The indication as to the possible source of the ear infection, although not the final proof, has been secured by testing serologically the strains of beta hemolytic streptococcus, of which cultures have been made simultaneously from the primary ear discharge and from the throat in the individual case. The results obtained in the nine cases studied with reference to this point show that the same serologic variety of streptococcus may be present in the throat and in the ear discharge.

In the majority of cases, cultures made from the ear discharge several days or weeks after paracentesis show that the apparent etiologic bacterium has become reduced in number, while the proportion of other varieties has increased.

Two cases have been encountered in which the beta hemolytic streptococcus has persisted in the ear discharge over a prolonged period. Further tests have shown that a different fermentative group of hemolytic streptococcus has appeared and finally supplanted the earlier variety. It is suggested that the presence of a second virulent variety may account for the prolonged duration of the disease in these cases.

Except for these two cases, the beta hemolytic streptococcus obtained in subsequent cultures from the ear discharge and from the throat in each individual case belongs to the same fermentative group as the hemolytic streptococcus isolated primarily from that case. Serologic tests carried out on the strains from nine cases show that all strains isolated originally and subsequently from a single individual case are similar.

In nineteen out of twenty-two cases of bilateral otitis media involved at approximately the same time, the same bacterial species has been obtained in the primary culture from both ears in each person.

Nine cases of bilateral infections yielding beta hemolytic streptococcus in the primary culture were studied. In eight cases, the strains isolated from both ears belong to the same fermentative group, and serologic tests carried out on the strains from four cases show that both ears have been infected with the same antigenic variety. One case was investigated in which the beta hemolytic streptococcus found in apparently pure culture in the right ear was of a different fermentative group than that occurring in the left ear. Since the original material was collected from the ear discharges several weeks after spontaneous rupture, no conclusions can be drawn in regard to the primary etiologic organisms.

The strains of beta hemolytic streptococcus from fifty-three primary ear discharges fall into the following fermentative groups: pyogenes, 45, or 85 per cent.; infrequens, 5, or 9.4 per cent.; equi, 3, or 5.6 per cent.

The serologic grouping by agglutination and agglutinin absorption tests of the strains of beta hemolytic streptococcus isolated from the primary ear discharges in forty-four cases of otitis media show the presence of three small serologic groups besides a large number of heterogeneous strains, among which no relationships have been found with the ten antiserums used. The first and largest group is made up of the strains from six cases; the second group consists of the strains from two cases, while those from the two other cases form the third group.

The heterogenicity of the strains isolated from the majority of acute ear infections during the period of increased incidence of respiratory disease in January and February, 1922, indicates the absence of a common primary or secondary causative agent.

From the data available, no deductions can be made concerning the condition of the tonsils in relation to the incidence and later course of the ear infections.

In the cases studied over a considerable period of time, diphtheroid bacilli and staphylococci have been found to be the most frequent secondary invaders in the ear discharge after the acute stage of the infection. There is some indication that in certain cases these bacterial varieties may be factors responsible for the chronicity of the inflammatory process.

The presence of numerous serologic varieties of beta hemolytic streptococci as the apparent etiologic organisms in a series of middle ear infections indicate that the use of a stock streptococcus vaccine is probably of little or no specific value. However, an autogenous vaccine made from the original variety in the ear discharge should be tried.

KENNEDY, Rochester, Minn.

TONSILLECTOMY AND SUBSEQUENT SUSCEPTIBILITY TO DIPHTHERIA AND SCARLET FEVER. JAMES A. DOULL, Pub. Health Rep. **39**:1833 (Aug.) 1924.

In a series of nearly 6,000 white children examined in the schools in Baltimore, chosen as a random sample from 25,000 children, 15.2 per cent. had had tonsils removed, the number increasing regularly from 6.9 per cent. at 5 and 6 years of age to 19.1 per cent. at 13 and 14 years.

Two hundred and twenty-four cases of diphtheria were investigated; of these, only two (0.9 per cent.) had had tonsils removed, though the expected number as judged by the previous figures was twenty-six; of 193 cases of scarlet fever, nineteen (9.8 per cent.) had had tonsils removed, whereas the expected number was twenty-five.

Children who had had tonsils removed were therefore distinctly less liable to diphtheria than those who have not had a tonsillectomy, while no significant difference in susceptibility to scarlet fever was shown.

SUTHERLAND, Chicago.

EFFECT OF TONSILLECTOMY ON THE GENERAL HEALTH OF TWELVE HUNDRED CHILDREN AS COMPARED WITH AN EQUAL NUMBER NOT OPERATED. ALBERT D. KAISER, J. A. M. A. **83**:33, 1924.

After presenting much valuable data, Kaiser says that one can attempt to draw some conclusions as to the value of tonsillectomy, but must not be unmindful of the dangers of the surgical procedure as well as the complications that do occur. One is impressed with the absence of any spectacular results when a group is studied controlled by a group not operated on.

Tonsillectomy offers a child considerable relief from colds, sore throats and mouth breathing. It lessens the chance of discharging ears and their complications. It assures some protection against glandular infection but is no guarantee against it and it does not assure the immediate disappearance of large cervical glands. It does not influence favorably or unfavorably infections of the larynx, bronchi and lungs. It does not prevent scarlet fever or measles but may influence the severity of the infections. It seems to lessen the incidence of diphtheria by removing fertile soil for the diphtheria bacillus.

It has not influenced the incidence of chorea or rheumatism but has shown a lessened incidence of heart disease over a period of three years. It has definitely reduced malnutrition in the group operated on as compared to the group that was not operated on.

HENN, Chicago.

APPLICATION OF BRONCHOSCOPE IN DIAGNOSIS AND TREATMENT OF AFFECTIONS OF THE CHEST. GEORGE E. MARTIN, Edinburgh M. J. Tr. Med.-Chirurg. Soc. **31**:129, 1924.

Martin remarks on the variety of cases reported, especially in the American literature, dealing with the removal of bronchial foreign bodies. He discusses the experience in Chevalier Jackson's clinic, which has been visited by himself and several of his colleagues from the British Isles. While British records show very few deaths from foreign bodies in the bronchus, the lay press occasionally reports deaths in children from inhalation of orange pips, safety pins, buttons and common playthings. Martin questions whether foreign bodies are less common in Scotland or whether it is not that many instances remain undiagnosed. In 1913, were reported by Turner and Fraser before this Edinburgh Society the successful removal of a small metal pipe from the bronchus of a boy, aged 7 years; a metal bolt from the right bronchus of a boy, aged 8 years; four months after swallowing a pin, the pin was successfully extracted from the bronchus of a boy, aged 4 years; a small bead was removed from the larynx of a child, aged 5 years; a whistle while being withdrawn from the respiratory tract of a child, aged 4 years, slipped into the esophagus and was swallowed.

The old classical signs are rare, the diagnosis largely clinical. Martin's conclusions are that there should be a roentgen-ray examination of the chest in suspected cases as well as in all instances of chronic bronchitis or of suspected phthisis; with non-opaque bodies the diagnosis may be overlooked. The speaker reviews the diagnosis as taught by Jackson and others and emphasizes the importance of careful search for these cases in his own country.

NEFF, Kansas City.

Diseases of the Skin

A SIMPLE METHOD FOR ESTIMATING THE LYMPHAGOGUE INDEX OF THE SKIN. FRANZ VON GRÖER, Ztschr. f. Kinderh. **38**:186, 1924.

A simple method is described whereby it is possible, with the intracutaneous injection of morphin chlorid from 1:1,000 to 1:1,000,000 to determine the lymphagogue index of the skin. The method includes a measurement of the wheal produced following injection of the morphin.

PETERMAN, Rochester, Minn.

Surgery and Orthopedics

NEPHROLITHIASIS IN CHILDREN, WITH REPORT OF A CASE. SIDNEY KLEIN, Arch. Pediat. **41**:505, 1924.

As a diagnosis in a pediatric clinic nephrolithiasis is a rare condition. The case reported by the author was the first patient with renal stone to be operated on in the last thirteen years at the clinic.

The child was a girl, aged 10 years, who had pain in the upper right quadrant, mild in character, for the last five years. It radiated across the upper abdomen to the left hypochondriac region. The pain was always worse at night and bore no relation to food or activity. Associated with the pain there was frequently nausea and vomiting. Headaches occurred almost every night. No dysuria, frequency, hematuria or fever. During the five years of illness diagnoses and treatment had always been directed to the gastro-intestinal tract. On physical examination the right kidney was found to be palpable and tender, fist percussion over the right lumbar region posteriorly caused pain. The urine showed a moderate trace of albumin, scattered white blood cells and an occasional red cell. This finding of an occasional red cell the author emphasizes as very important in the diagnosis. Roentgen ray revealed shadows of one large stone and three smaller ones in the region of the pelvis of the right kidney. At operation the large stone was removed but no trace of the smaller ones was found. After operation the roentgen-ray shadows of the smaller ones still persisted.

VON MEYSENBUG, New Orleans.

TREATMENT OF EMPYEMA IN CHILDREN. JOHN FRASER, Edinburgh M. J. Tr. Med.-Chirurg. Soc. **31**:145, 1924.

Fraser apologizes for considering such an old subject on the basis of getting out of the rut, and discusses the time and choice of methods of pus removal from the thoracic cavity. In the surgical wards of the Children's Hospital, empyema is rather rare, fifteen out of each 1,000 surgical cases yearly. He is interested in determining how often acute lung affections result in empyema, reporting only hospital cases: empyema follows acute lobar pneumonia in 21 per cent., influenzal pneumonia 24 per cent., bronchopneumonia in 54 per cent. The factors depend, of course, on age and general health, but his figures are parallel with those of Mounetier. That bronchopneumonia is more frequently

followed by empyema is contrary to the usual conception, but as bronchopneumonia is more diffuse, it is possible that the barriers are less complete and the infection more easily reaches the surface around the fissures.

In the fifty-three pneumococcal cases the mortality was 6 per cent., and in the seventeen streptococcal cases the mortality was 24 per cent.; in the former the effusion is limited by dense and extensive adhesions, the streptococcal effusion is widespread and the adhesions few. The presence or absence of adhesions influences the kind of treatment, and these are determined by the estimation of the pleural tension, occasionally aided by roentgen-ray lateral projection with the aid of the Potter-Buckey diaphragm. Pleural tension is determined by an aspirating needle-rubber tube connection with a water manometer, which shows in the absence of adhesions a wide play in the respiratory oscillations, varying from a negative 5 to a positive 10. When the visceral and parietal surfaces are adherent extensively, a constant, little varied pressure is maintained. A good general condition of the child is another factor which permits of more extensive surgical relief. When the effusion is circumscribed by adhesions, usually a pneumococcal infection, the ideal treatment is a free opening, by resection or intercostal incision, evacuation of the pus and masses of fibrin, closed drainage at the lowest point, disinfection of the edges and closing of the original wound, with healing in twenty-one days. Details of the operation are given. In the nonadhesive group (streptococcal) free incision with extensive collapse of lung is disastrous. Aspiration of the usually thin fluid is performed for four or five days when developing adhesions allow the chest to be opened with considerable safety. Simple thoracotomy with opened drainage resulting in pneumothorax, mixed infection and chronic sinus has many objections. Irrigation with dye and other solutions has shown no advantages. Martin suggests the adjuvant action of internal administration of acid sodium phosphate.

NEFF, Kansas City.

SUBMAXILLARY ADENOPATHIES OF THE TYPE OF SCROFULODERMA TREATED WITH THE QUARTZ LAMP. BARRIO DE MEDINA, Arch. Españ. Pediat. **8**:221, 1924.

The author reports very favorable results from the use of the quartz lamp in the treatment of a case of submaxillary adenopathy with broken down glands.

Eleven exposures were given, the first one of ten minutes' duration with the lamp at a distance of 30 cm. The duration of exposure was gradually increased to twenty minutes and the distance decreased to 20 cm.

The lesions showed rapid improvement, with complete disappearance of purulent secreion and of fungous tissue, and eventual healing with smooth cicatrix.

SCHLUTZ, Minneapolis.

EXSANGUINATION-TRANSFUSION. L. BRUCE ROBERTSON, Arch. Surg. **9**:1, 1924.

The author reports the results of 160 blood transfusions in children to combat the toxemia of severe burns, erysipelas, acute intestinal intoxication, resorcin poisoning, malignant scarlet fever and septicemia. Blood was removed by the citrate method from several donors in 100 c.c. amounts until sufficient quantity was obtained to more than make up for the total volume of blood in the body of the child to be transfused. Blood was removed from the child either at the fontanel or femoral vein until the initial signs of exsanguination appeared, when the citrated blood was allowed to enter at the median basilic or long saphenous vein. This process was continued until a complete interchange of blood had taken place. In fourteen patients with severe extensive burns of the body surface, all of which looked helpless, seven recovered. In six cases of erysipelas in the new-born, which ordinarily are always fatal, three children recovered, and in fifteen ranging from 1 to 12 months of age, three patients recovered. Forty infants suffering from acute intestinal

intoxication were transfused resulting in seventeen deaths. In eight of these patients which appeared to be dying, four recovered, while in six similar patients treated medically only one recovered. In two infants suffering from resorcin poisoning as a result of too strong an ointment being applied for eczema, transfusion resulted in recoveries after both looked hopeless. In five cases of malignant scarlet fever, with the patients in coma, all made rapid recoveries. In septicemia, transfusion was used four times with two recoveries.

MILLER, Chicago.

Miscellaneous

HELIOTHERAPY. R. PLATO SCHWARTZ, Boston M. & S. J. **191**:243 (Aug. 7) 1924.

Method of using heliotherapy and other measures in the treatment of osteo-articular tuberculosis at the J. N. Adams Memorial Hospital is described in detail. As to the results the author says: "Patients with a tuberculous bone or joint, systematically treated according to the above outline, are greatly improved in general health, while the extremity frequently regains useful function within eighteen months to two years, when ankylosis in a favorable position was the best the past could offer."

GENGENBACH, Denver.

A STUDY IN PREVENTIVE AND SOCIOLOGICAL MEDICINE. MARK S. REUBEN, Arch. Ped. **41**:435, 1924.

A detailed study of the economic, medical and social problems of twenty-one families in the district surrounding the College of Physicians and Surgeons in New York City is described. A trained social worker carried on the work, visiting each family at least every six days, gaining their confidence and cooperation. Medical examination and treatment was given to every member of each family and financial aid secured when needed. Acute illnesses were attended by the Vanderbilt Clinic physician. The most striking impression made in the survey is that the problems of 10th Avenue are the problems of Park Avenue and Riverside Drive. There are no problems peculiar to this type of neighborhood. The problems of 10th Avenue are the problems of humanity everywhere.

VON MEYSENBUG, New Orleans.

HYPOPLASIA OF THE ENAMEL OF TEETH AS AN INDEX OF DISEASE DURING THE FIRST YEAR OF LIFE. KARL KASSOWITZ, Ztschr. f. Kinderh. **38**:225, 1924.

Deformities of the dental enamel can be traced according to their location to a developmental or growth disturbance at a corresponding age. The greatest disturbances are produced by acute, severe, febrile infections, measles, pneumonia, and pertussis (in that order). Rachitis, tetany, syphilis, and tuberculosis play a rôle, but one not so important. Although the dental hypoplasia indicates a definite, severe, developmental injury at a certain early age, it is not a residuum of any specific disease.

PULSE FREQUENCY AND SITTING HEIGHT. CLEMENS PIRQUET, Ztschr. f. Kinderh. **38**:301, 1924.

Two hundred twenty-six new-born, twenty infants in the first year, and seventy-two children from 1 to 14 years of age were studied. The pulse frequency, duration, and the energy exchange value are measured and the relations to the heart weight, body weight, and sitting height are computed.

PETERMAN, Rochester, Minn.

LIFE TABLES FOR THE POPULATION OF NEW YORK STATE ACCORDING TO NATIVITY. J. V. DEPORTE, Am. J. Hygiene **4**:302 (July) 1924.

A comparison of the life tables for white males, aged 20 and over, of the three large divisions of population of the state of New York, namely, native born of native parentage, native born of foreign or mixed parentage, and foreign born, based on the average numbers of deaths in the years 1909, 1910 and 1911, as given in the U. S. Mortality Reports, and the population, as given in the U. S. Census Report, shows that:

(*a*) The death rates of native born white males of native parentage has been lower and the expectation of life higher than of either the mixed or foreign group at practically every year of the life span.

(*b*) That the death rate of foreign born white males has been lower than those of the mixed group, from the twentieth to the fifty-eighth year, and from the seventy-fifth year on. Between the ages 58 and 75, the death rate of the mixed group had been lower than those of the foreign group.

(*c*) One-half of the original number, 100,000 of white males, among native born of native parentage, reach the seventieth year, among native born of mixed or foreign parentage the sixty-first year, among foreign born the sixty-third year.

There are several reasons for more favorable conditions among foreign born as compared with the native born of foreign and mixed parentage:

1. Immigrants are usually above the physical average in their homeland.
2. Restrictive legislation has barred physical and mental defectives, convicts and paupers from entering the country.
3. The influence of occupational differences.
4. Mainly, because the native born of foreign parentage are unable to adjust themselves to their new social surroundings.

SCOTT, Pittsburgh.

THE MORTALITY OF THE CHINESE IN THE UNITED STATES, THE PHILIPPINES AND HAWAII BY CAUSE OF DEATH. C. E. A. WINSLOW and Z. W. KOH, Am. J. Hygiene **4**:330 (July) 1924.

A study is made of the comparative death rate of Chinese in the United States, Hawaii and the Philippines for the purpose of finding an explanation for the generally high mortality of the Chinese in the United States.

Analysis of deaths by cause indicate that the Chinese in all three regions studied exhibit an excessive mortality from tuberculosis, circulatory diseases, and genito-urinary diseases, as compared with Caucasians. It would appear that inherent characteristics or racial habits must exert a specific predisposing influence. The excessive mortality from acute respiratory diseases in the coast states is apparently due to local climatic or social conditions, since death rates from these causes are not excessive in Hawaii or Manila.

MATERNAL AND INFANT MORTALITY IN PHYSICIANS' FAMILIES. HAVEN EMERSON, Am. J. Hygiene **4**:365 (July) 1924.

In order to determine the relation existing between the general maternal and infant death rate and that in physicians' families, the author sent out a form letter, to which he received 751 replies, involving 1,974 pregnancies and 1,910 living children. A summary of the data thus obtained shows that the maternal risk rate is 45.5 per 10,000 pregnancies, as compared with a general maternal risk rate of 81.4. Also that the infant mortality rate is 43.5 per 1,000 births as compared to 79.6 per 1,000 births as shown in the death registration area of the United States for 1922.

If the maternal and infant mortalities could be reduced throughout the population to the rates which seem to prevail among the families of physicians in the United States, 4,000 lives of mothers and 11,000 lives of infants under 1 year of age would have been saved in the year 1922 alone.

PRICE, Pittsburgh.

THE RESULTS OF A QUESTIONNAIRE IN REGARD TO POSTENCEPHALIC INJURIES IN CHILDREN. J. B. JOERGER, JR., Schweiz. Med. Wchnschr. **54**:679 (July 24) 1924.

The article is based on a questionnaire sent out by the Swiss Psychiatric Society to every Swiss physician to ascertain the number of persons suffering from postencephalitic injuries and in each case the degree and the nature of severity. This was done as a means of considering a recommendation of Professor Maier-Burghölzli to establish an institution with the especial purpose of taking care of these unfortunates. Some thirty physicians responded to the query. Seventy-seven cases were cited.

The author reviews the mental abnormalities which are apt to follow epidemic encephalitis (lethargica) dividing them into two groups: (1) the mentally active type which manifests moral delinquencies, (2) the apathetic type which is stolid and excessively sluggish in mental reaction. He discusses several cases which have come to his personal attention as examples. He heartily endorses Professor Maier-Burghölzi's asylum suggestion.

ELTERICH, JR., Pittsburgh.

THE DEATH RATE OF HONG KONG AS A CRITERION OF MORTALITY IN CHINA. LI TING-AN, Nat. M. J. China **10**:25, 1924.

No Chinese city keeps accurate vital statistics, according to the author, and this study is an attempt to utilize Hong Kong's death rates to get a general idea of the subject. Hong Kong is more progressive than most Chinese cities and has prevented many controllable deaths. Tables are given for deaths at different ages, for all causes, for tuberculosis particularly, and comparisons made with other countries.

A true infant mortality of Hong Kong cannot be calculated because of the lack of accurate birth registration, and only the mortality under 1 year of age is used. The infant death rate of Chinese children in Hong Kong in 1922 was 289.98 per 1,000. The standardized death rate of the Chinese in Hong Kong for the same period was 27.57 per 1,000. The infant death rate of the Chinese is several times larger than the infant mortality of many other places cited, though it is less than current opinion would have it.

Li Ting-An believes his figures indicate approximately the mortality rates which will be found in other South China cities, when accurate statistics are available, except for years of epidemics.

LUCAS, San Francisco.

THE OVER-WEIGHT CHILD. BORDEN S. VEEDER, J. A. M. A. **83**:486, 1924.

Veeder discusses the overweight child from the standpoint of ordinary obesity in which there is a generalized distribution of the excess fat tissue with no evidence of endocrine disorder. He considers heredity a factor and perhaps here it is that environment plays its rôle. With obese parents, the child usually sits down to a table overloaded with foods of high caloric value. The obese child is usually as bright mentally as the child of average weight.

Treatment is based on exercise and food. The diet of the obese child should contain large bulk with low value. Patience, cooperation and perseverance over long periods of time are the essentials in treatment.

HENN, Chicago.

Book Reviews

CONTRIBUTION TO THE STUDY OF MYOCARDITIS IN CHILDHOOD WITH PARTICULAR REFERENCE TO ISOLATED ACUTE MYOCARDITIS AND TO THE POSSIBLE RELATION OF MYOCARDITIS TO STATUS THYMICOLYMPHATICUS. By MATTHEUS VISCHER, Berlin, Verlag von S. Karger, 1924.

The author, in this monograph, discusses the subject of myocarditis in childhood, with particular reference to two phases: the occurrence of isolated acute myocarditis, and the possible relation of the status thymicolymphaticus as a predisposing factor or actual factor in the development of this condition.

His conclusions are based on careful observation of material from the pathologic anatomic Institute of Basel, and comprises 793 necropsies, of which 530 were on children ranging in age from birth up to 15 years, and 263 on prematurely born infants. In practically all of the material microscopic examination of the heart muscle was made.

Status thymicolymphaticus was definitely established in only thirty-two cases. The criteria on which the diagnosis of this condition was based consisted in definite and unmistakable enlargement, or hypertrophy, of the thymus gland associated with general enlargement of all lymphatic glands and structures, including hypoplastic changes in the suprarenals. Interstitial infiltration of the myocardium, inflammatory or otherwise, was found in twenty-seven cases. In only four cases was myocarditis associated with a status thymicolymphaticus.

An exhaustive and critical review of the entire literature covering this subject, or anything bearing on it, is given. This is particularly true of the reports and conclusions of Ceelen, Fahr and Riesenfeld.

Every conceivable form of interstitial myocarditis and causative factors responsible for it is mentioned or discussed.

This is supplemented by a most complete and detailed account of the author's own twenty-seven cases, with critical comment on the underlying causes of the condition.

In addition, he reports from his necropsy material five cases of isolated acute interstitial myocarditis, in which there was no assignable cause. The trouble seems to begin as a primary lesion in the myocardium.

The author concludes from his studies that myocarditis does not occur as the result of constitutional anomaly, or have any connection whatsoever with the condition known as status thymicolymphaticus. Cases of myocarditis observed in connection with this constitutional condition are merely an accidental association.

The cause of primary acute interstitial myocarditis remains obscure.

It is also evident that this study, splendid and exhaustive as it is, really sheds no additional light or offers any further explanation for the development of the peculiar constitutional anomaly known as status thymicolymphaticus.

STRAHLENBEHANDLUNG BEI KINDERKRANKHEITEN. By VON PROF. DR. W. BIRK, Vorstand der Kinderlinik in Tübingen, and DR. L. SCHALL, Assistent der Kinderlinik in Tübingen. Urban and Schwarzenberg, Berlin and Vienna, 1924, 77 pp., illustrated.

According to the authors this book is a critical discussion of the uses of the quartz lamp and the roentgen ray in the therapy of diseases of children. Since the senior author is one of the leading German pediatricians it should be a valuable contribution to medical literature.

The text apparently bears out the authors' contentions. It is divided into two parts, the first dealing with quartz lamp therapy and the second with the roentgen ray. The work is based mainly on the authors' observations, but there are many references and a good bibliography. They discuss to a varying degree, etiology, symptomatology and diagnosis while the specific therapy is described in detail. No extravagant conclusions are drawn.

In both the first and second parts there are short articles on the ill effects which sometimes follow the treatments.

Most of the space in the first part is given to the discussion of rickets, spasmophilia and tuberculosis. They consider the quartz lamp a distinct addition to the present treatment of rickets. Short prophylactic exposures, three times weekly, are recommended for artificially fed infants during the autumn and winter months. They were unable to prevent rickets in all infants in an asylum by the administration of two teaspoonfuls of cod liver oil daily, but with cod liver oil plus the ultraviolet rays no rickets occurred. Some of the statements on the subject of rickets would hardly be accepted by American pediatricians. For example, on page 3 the authors state that rickets occurs only in the winter, and if it is ever present in the breast fed infant it is confined to the skull (craniotabes).

Nine different forms of tuberculosis are considered. They find the quartz lamp especially valuable in tuberculous adenitis and the exudative type of peritonitis. They take an unusually pessimistic attitude toward pulmonary tuberculosis in children. On page 19 the following sentence is found: "It is a peculiarity of childhood, that tuberculosis, when it has involved the lungs to the extent of producing clinical signs, is no longer curable."

The itching of chronic eczema in infants is often greatly benefited by quartz lamp exposures, although it may become worse after the first one. In asthma and nocturnal enuresis a few good results were obtained which, they think however, may have been due to the suggestive element.

Roentgen-ray technic is described in considerable detail, yet simply enough so that the average physician could grasp most of it. Tuberculosis, thymic hyperplasia, diseases of the blood and spleen and eczema are the chief indications for this type of therapy. There is a very full discussion, with plates on enlargement of the thymus.

This treatise, we believe, is a very fair and conservative opinion of the uses of these valuable therapeutic appliances in the treatment of sick children.

THE TREATMENT OF PULMONARY TUBERCULOSIS IN CHILDREN BY MEANS OF ARTIFICIAL PNEUMOTHORAX. By H. ELIASBERG and P. CAHN. Abhandlung aus der Kinderheilkunde und ihren Grenzgebieten, Beihefte zum Jahrb. f. Kinderh. Part 1, 1924. Berlin, S. Karger.

Of all the procedures which have been advocated for the treatment of pulmonary tuberculosis, probably none has equalled in daring and originality the proposal to produce an artificial pneumothorax on the affected side. Now widely accepted as a valuable therapeutic aid in certain types of cases in adults, its use in children has been much less general, and still more or less in the experimental stage. The present monograph, the first of the new supplements to the Jahrbuch für Kinderheilkunde, is especially timely therefore, containing as it does a very complete discussion of the technic, indications for the operation, complications, immediate effects and end-results obtained in 125 children with tuberculosis in whom this procedure was employed. The observations reported cover a period of about ten years, and it is interesting to note that forty-one of the children were less than 3 years old. Resorted to at first only in the one-sided ulcerated or cavernous type of case, its use was extended as the apparent harmlessness of the procedure was demonstrated (no deaths due to the injection of air in ten years) until finally the authors included cases of severe hilum tuberculosis and in four children produced a

pneumothorax of both sides simultaneously. Using manometer readings of the intrathoracic pressure and the roentgen ray as the chief controls, the authors believe the method is harmless and of considerable value in the treatment of tuberculosis in children. Regarding the latter phase of the work, however, the figures are not entirely convincing: Only fifty of the children had bacilli in the sputum at the time of the treatment and of these, 68 per cent. died, whereas only six of the remaining thirteen children are at present without fever or cough. As might be expected, the results were better in 61 "closed" cases (without bacilli in the sputum); 14.7 per cent. died, 73.8 per cent. are living and in 11.4 per cent. the fate is still uncertain. Other methods of treatment would no doubt compare favorably with the last figures, but despite this fact there is probably a distinct field of usefulness for artificial pneumothorax in childhood. The article is a well written, thorough presentation of a series of careful observations and should by all means be read by everyone interested in the treatment of tuberculosis in childhood.

Correspondence

"ROENTGEN-RAY STUDIES OF STOMACH FUNCTION"

To the Editor:—My attention has been called to a misinterpretation in one of my references to the German literature on "peristolic function." Having reviewed the paper in question, I wish to correct the inaccuracy, which however accidental, is apt to mislead.

In the confusion resulting from definite differences of opinion concerning the actual existence of a peristolic function, Flesch-Peteri have been misinterpreted as being among those who are not convinced of its existence.

In my first paper on stomach function I have written; "Leven and Barrett think that the consistency of the food plays the greatest rôle in the filling and form of the stomach. Flesch-Peteri agree with this point of view and add that the quality of the feeding is of distinct influence on the development of the form of the gastric organ" (*Am. J. Dis. Child., July*). This in general, is a correct statement of their belief.

In a second paper, devoted to the peristolic function, I mentioned: "Flesch-Peteri, who also claim that, according to their observations, it is impossible to demonstrate a peristolic reflex" (*Am. J. Dis. Child., July*). Now this statement corresponds exactly with their assertion: "In the infant stomach there is no trace of this peculiarity" (meaning peristolic function) (*Ztschr. f. Kinderh.* **2**:280, 1911). However, it appears, later in their work, that these authors meant to say it is impossible to demonstrate a peristolic reflex with fluids!

The interesting point, which Flesch-Peteri bring out, and which was not mentioned in my paper, is that the stomachs of infants under the age of 1 year, seem to differ from the stomachs of children over 1 year, in that the former show no peristolic function with a normal diet while the older children do. Their final explanation is that a normal infant's diet is fluid, while the normal diet of an older child includes cereal. Since the peristole can only be elicited with cereals, or other thick foods, it does not appear with the normal, fluid diet of infants until their food is thickened. Thus, with a proper feeding the peristole appears in the stomachs of infants and older children alike, the original point of differentiation having been removed.

I have also quoted Lindberg as successively denying and affirming the existence of a peristolic function. He corrects this impression, however, in a letter explaining that, in the first place, he is only referring to the literature. Secondly, he confirms the opinion of Flesch-Peteri and further explains how the absence of a proper peristolic function in the stomach of an infant lying in the horizontal position accounts for a type of habitual vomiting and how this can be corrected by holding the child in a vertical position. It is not difficult to confirm the practical importance of this observation.

JULIAN L. ROGATZ, M.D., New York.

CORRECTION

The article by Drs. Burhaus and Gerstenberger, entitled "The Protective Power of Infant's and Maternal Serum Against Pneumococci," published last month as having been read before the American Pediatric Society, was not presented before that society.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, May 20, 1924

H. W. Cheney, M.D., *President*

The Development of the Teeth. Presented by Dr. Arthur D. Black.

The Deciduous Teeth.—The deciduous teeth begin their formation at about the seventeenth week of uterine life, and the crowns of the twenty deciduous teeth are practically complete in their formation at birth, so that malformation of the deciduous teeth, such as often presents in the permanent teeth as a result of disease in childhood, is not likely.

The roots of the deciduous teeth are not formed until several years after the crowns are completed, on the average of from two to three years afterward. Therefore, the roots are not completed at the time the teeth are erupted, but usually a year or a year and a half after eruption. If decay penetrates the dentin and involves the pulp of the tooth, so that the pulp dies before the completion of the root, then root formation stops. An abscess forms at the apex of the root and the tooth must be extracted, because it is impracticable to treat a tooth while the root is only partly formed. This applies to both the deciduous and the permanent teeth.

The roots of the temporary teeth begin to be absorbed, on the average, about three years after they are fully formed, and absorption continues to the time that these teeth are shed. The absorption progresses as the crowns of the permanent teeth gradually develop and approach their time of eruption, and also approach the surface of the gum as the roots of the deciduous teeth gradually disappear.

If the pulp of a deciduous tooth dies and remains untreated, and an abscess develops, the presence of infection about the root apex is likely to delay the absorption of the root. In such cases the corresponding permanent tooth must usually erupt in an abnormal position, because it is deflected from its natural route through the gum by the unabsorbed root of the deciduous tooth.

FIRST PERMANENT MOLARS

I suggest that you should call to the attention of mothers of children under 6 years of age the fact that each of the four first permanent molars erupts back of the deciduous teeth, without a deciduous tooth being lost for the first molar to take its place. There should be, under normal conditions, at 6 years of age, twenty-four teeth in the mouth, twenty deciduous and four permanent teeth.

This is important, because an enormous number of first permanent molars are so badly decayed that they must be extracted, and in the majority of cases the parents have no idea that they are permanent teeth, because no deciduous tooth has been lost. The first permanent molar usually erupts without discomfort to the child, and its eruption may be unnoticed by either child or mother. On the other hand, the deciduous molar teeth usually erupt with considerable difficulty; often the child is quite sick. The first permanent molars erupt at about the age of 6 years, although I have seen all four of the first permanent molars in full eruption in a child a few months beyond his fourth birthday, so that we have wide variations of the time of eruption, but the first permanent molar is more likely to erupt earlier than the sixth year, rather than later.

PERMANENT TEETH IN TWO GROUPS

The permanent teeth, from the standpoint of development, are to be classified very definitely into two groups, the first permanent molars and the six front teeth in one group; the bicuspids and the other two molars in the other group. This is an important classification from the standpoint of development, because in the case of this first group (the first permanent molars and the six front teeth), we will often see very definite effects of disease or malnutrition during childhood, which interferes with the normal functioning of the tissue elements. We may observe defects in this group of teeth, the position of the defect corresponding to the period of an illness or malnutrition, occurring during the first five years of life.

The first permanent molars begin the formation of their enamel at birth, sometimes a little earlier. The cusps of this tooth are formed first and the remainder of the crown to its junction with the root is formed at about the end of the fourth year. The apex of the root is completed almost any time between the ninth and the fourteenth year, so that a period of from 9 to 14 years is required for the formation of the first molar teeth. If a decay of this tooth occurs, which exposes the pulp and causes the pulp to die before the roots are fully formed, it becomes necessary to extract the tooth, because the root canals cannot possibly be filled.

The edges of the central incisors begin to be formed at about the first year, the lateral incisors at about the second, and the cuspids at about the third year, and it requires four or five years for each crown to be completed. Therefore, lines drawn to indicate the periods of contemporaneous calcification of this group of teeth would, for any given period, be nearer to the gum on the first molars, slightly farther away from the gum for the central incisors, still farther for the lateral incisors, and fairly close to the points of the cusps of the cuspids.

HYPOPLASIA OR ATROPHY OF THE TEETH

The effect of diseases of childhood on these teeth, if there is any effect, will be seen as pits, grooves, or irregularities of formation in horizontal lines around all of those teeth that are in process of calcification at the time of the illness. This condition is called hypoplasia or atrophy of the teeth. A serious illness, or a period of malnutrition does not necessarily cause defects in the teeth, but in an unknown percentage of cases there are defects in formation of both enamel and dentin.

We may judge most definitely from the central incisors and the first molars as to the year of the particular illness which caused this defect. If a defect is noticed in the upper central incisors, in which we are most likely to notice the malformation, there will always be a corresponding defect of the first permanent molars, and very commonly the defect of the first permanent molars is much more serious than that of the incisors. It is not only a more serious defect in the actual structure of the enamel, but also because of the greater likelihood of the first permanent molars to decay.

SO-CALLED HUTCHINSON TOOTH

The so-called Hutchinson tooth, which has been described in the medical literature ever since the time that Hutchinson first claimed that this particular defect was indicative of congenital syphilis, is familiar to most investigators. This particular defect, which is the result of malnutrition or illness during the first 18 months of life, involves the edge, the entire crown of the tooth being as a notch at the center of the edge, the entire crown of the tooth being slightly peg-shaped, or narrower at the cutting edge than it is at the gum line.

The enamel which forms the edge of this tooth is calcified from three points of beginning, forming three lobes. The central lobe begins its formation first, so that if there is a period of definite malnutrition after birth which extends

beyond the first year, the edge of the central lobe may be malformed and the two lateral lobes are drawn in over the central portion, so that the tooth is narrower at the incisal edge, with a notch in the center.

The point that I should like to make in this connection is this: Without question, we do find this condition, the Hutchinson tooth, in the child of a syphilitic mother, yet exactly the same defect may be present in the teeth of a nonsyphilitic child, resulting from illness or malnutrition occurring at the time when this enamel is being formed. The most positive indication that the Hutchinson tooth results from malnutrition lies in the fact that the four first permanent molars, which are also in process of calcification are always malformed in every mouth in which we find a so-called Hutchinson tooth. This defect does not, therefore, always point to syphilis.

The six front teeth and the first molars are considered as a group; hypoplasia of any of the other teeth very seldom occurs. In other words, a disease or period of malnutrition which occurs after the fifth year seldom results in this particular defect of the teeth, so that the bicuspids and the second and third molars will not show this defect.

MOTTLED ENAMEL

There is one other peculiar condition that we might mention in this connection. There are some twenty-eight different areas in the United States, mostly in the west and southwest, and particularly in the city of Colorado Springs, in which there is a condition of dystrophy that is referred to as "mottled enamel." Whenever any of you are in Colorado Springs you may notice that the incisor teeth of a majority of the residents are more or less blotched. They may be in part pure white, the blotches very irregular in form, or they may be yellow, or brown or jet black. They may be any color that might be obtained from the coloring elements of the blood. Eighty-five per cent. of the children who were born and lived in certain sections of Colorado Springs up to their fifth year, have blotches on the teeth of this particular group.

Efforts are being made to learn the cause of this peculiar endemic condition. It was first thought to be the drinking water but during the period of this study the source of the drinking water of Colorado Springs was entirely changed. On one of the rivers of Arizona, where there are two Indian reservations, one tribe living on one side of the river and another tribe living on the other side, and both taking their drinking water out of the same river, it was observed that in one tribe not a single tooth could be found mottled, while in the other tribe 98 per cent. had mottled teeth.

There is one difference in the occurrence of mottled enamel as related to hypoplasia. If a child is taken into one of these endemic regions after the fifth year, when the enamel of the teeth of the first group has been formed, there is likely to occur a blotching of the bicuspids and the second and third molars. In other words, teeth that are in process of formation in the jaws of children during their residence in one of these areas are 85 per cent. likely to have these blotches.

RELATION OF EARLY CARE OF TEETH TO THE GENERAL HEALTH

In finishing, I should like to speak on the question of the relationship of the early care of the teeth to the general health. There are three related things that come to mind which all of you ought to consider in handling children. If we were to make an examination of the teeth of all the children in the grades of our public schools on any one day, we would find an average of about seven cavities to each child, more cavities than all of the dentists in Chicago could possibly take care of if they worked on none but those children. Let us relate this to a radiographic study of the mouths of some hundreds of adults, all of whom were apparently in good health and attending regularly to business. This showed 78 per cent. to have one or more areas of destruction

of bone of the mandible or maxillae about the teeth; 53 per cent. of them had one or more abscessed areas where bone had been destroyed at the root ends of the teeth; and 55 per cent. had one or more pyorrhea pockets that had gone far enough to destroy some of the bone along the sides of the roots of the teeth. Some had one and some had the other, and some had both. Putting the two groups together, 78 per cent. of these presumably well persons had one or the other or both types of chronic infection about the teeth.

Let us think of these things in relation to systemic disease in later life. These three things are very definitely related, and the only way that the dental profession is possibly going to catch up and handle its part of the problem of focal infections is not by continuing, as we are now, to extract untold thousands of teeth every year, because there is infection about them. We must get back closer to the source, and pay more attention to the teeth of the young child, and especially the teeth of the temporary set and the first permanent molars.

Dental caries should be classified as one of the diseases of childhood, because the average person is very susceptible to dental caries in childhood, and much less so with advancing years. This compares with other diseases of childhood. Some children are very susceptible and catch almost every disease that comes along, while others seem to be very nearly or quite immune. There is a difference in susceptibility in children to dental caries, although not so marked as to other diseases. They gradually gain immunity as the years go by, and this only emphasizes the more the importance of the best of care during the childhood period.

TREATMENT INDICATED IN CHILDHOOD. Presented by DR. F. BLAINE RHOBOTHAM.

The word prophylaxis, as is generally used by dentists and the laity, refers to cleaning teeth. Some of us like to think of it as meaning many things other than cleaning teeth; in fact, an effort to prevent disease.

The very first beginning of prophylaxis, in the way of teeth, the deciduous teeth, should begin before birth, because the deciduous set of teeth begins its development about the sixteenth week of fetal life. Hypoplasia or atrophy is frequently seen in deciduous teeth. I very frequently see teeth of children under 2 years having little or no enamel. It is hard to diagnose these conditions. The dentin, when it is not covered with enamel, may appear decayed when it is not. If it is not decayed, it can be treated sometimes successfuly with the application of a strong silver nitrate solution. This solution should be applied at frequent intervals to protect the exposed surfaces. Sometimes silver nitrate is applied to those surfaces in error. I mean by that that the dentin may be diseased with caries, and as silver nitrate does not penetrate very deeply the decay may go on even after its application.

When the enamel is faulty or lacking, or the normal contour and dental anatomy of the tooth is at fault, many times a great deal can be done in the way of prevention by grinding away the rough edges of enamel, making the tooth as smooth as possible, so that débris will not adhere to it readily. Then the use of silver nitrate where it is indicated is in order.

If there is a very thin layer of dentin, the silver nitrate may penetrate so deeply that it will cause hyperemia of the pulp of the tooth, which results in very serious pain, and often in the death of the pulp itself.

The use of black copper cement as a preventive measure in protecting teeth, this applies to the six year molars as well as to the deciduous teeth, is a sort of tricky proposition, too. Black copper cement will not prevent caries from progressing. The fault and mistake of such treatment is very common, that is, of using black copper cement in the grooves of the six year molars where there is initial caries. However, if there are no caries, black copper cement in the grooves of the six year molars, in rough places, in grooves and in fissures on the deciduous teeth, will act as a protecting agent against caries.

The question is often asked, when should one begin cleaning and caring for the child's teeth. The average parent begins much too late; sometimes they fear they will destroy the enamel body by brushing the teeth. They think that the enamel is not so hard as it will be a little later on, and that it is new and soft enamel as they call it, and that it can be easily destroyed by any abrasive used in a tooth paste, in a tooth powder or by the brush itself. That is an error. As soon as the teeth erupt they need hygienic treatment.

How often should a child be seen by a dentist for examination will have to be established by those examining the child. Some dentists call their patients in every month, others every three months, others six months, and some every year. Any one of those intervals may be sufficient and right in one case, and it may be entirely wrong in another.

You may find that the period changes, that is, there are certain ages of childhood when they are more susceptible to caries than at others. The age is not constant with persons. I have seen twins receiving the same hygienic treatment of the teeth and care of their mouths, in which one child had a lot of caries and the other no caries at all. I have seen children that had had no hygienic treatment, with no caries; I have seen children receiving a lot of hygienic treatment with a number of caries. That is no argument against hygiene of the mouth, but it only proves that, in spite of hygiene of the mouth, sometimes we are unable to cope with disease. However, if a parent is faithful and the dentist is conscientious, the situation can be kept pretty well in hand.

The first place that caries attack is the anterior teeth, and fortunately so, because they are the first teeth to be lost. Usually the eight anterior deciduous teeth are lost before the child is 8 years of age. The ages vary a great deal with children. I have noticed that children of southeastern Europe, and of Jewish descent, lose their teeth earlier and the developing permanent teeth come in earlier.

Initial caries in the anterior teeth of the deciduous set can usually be treated without fillings. The proximal surfaces of the teeth can be ground smooth and flat, at the expense of the lingual angle of the tooth, and the dentin surfaces treated with silver nitrate. There is a natural spreading of the anterior teeth at about the third, fourth and fifth year. As a result of that spreading this treatment can be used without any fear of interfering with development, because the teeth are naturally separated at that time. With teeth in the posterior part of the mouth, conditions are different. If caries begin in the proximal surface of those teeth, contacts should be preserved for purposes of development.

It is very difficult to fill proximal cavities in the deciduous lower teeth, because the dental anatomy of the deciduous molars is slightly different from the dental anatomy of the permanent molars, in that the deciduous molars have more enamel and less dentin on the proximal surfaces as compared with the corresponding permanent teeth. As a result, if caries go through the enamel on the proximal surfaces of the deciduous teeth and reach the dentin, there is very little depth in the dentin for cavity preparation. If there is no definite cavity preparation, the fillings do not stay well in place.

Many parents as well as dentists become discouraged about fillings in deciduous molars, because they do not stay in. The fault is that the child is treated too lightly in the dental office. Cavity preparation in the deciduous molars should be as carefully done as it is in permanent molars. There must be a retention form for the filling. Care must be taken that the pulps are not exposed, that the filling materials used are such that will last. The permanent deciduous molars usually last until the child is from 9 to 11 or 12 years of age. If a deciduous molar is decayed at the age of 6, a cement filling will not last as long as the tooth does, nor will any other sort of a temporary filling last. If these teeth are to be watched, they should be filled with such fillings as are permanent. When placing permanent fillings in teeth, such as those made of silver amalgam, there is always danger of causing death to

the pulp of the tooth. That is another thing that discourages parents in having the children's teeth taken care of, for if a large metallic filling is placed close to the pulp of a deciduous tooth it may result in death to the pulp of that tooth from thermal changes passing through the filling. If black copper cement is used (and it is used very extensively) too near to the pulp of the tooth, it is irritating to the pulp. The most severe type of toothache results from the use of black copper cement in deep cavities, or in cavities approaching the pulp of the teeth.

If a lasting hyperemia is started up in the pulp of the tooth, and the irritation continues, the pulp will die. Sometimes the pulps of deciduous teeth die, just as they do in permanent teeth. I mean by that they sometimes die without pain, and if they die without pain the next time that the child is observed by the parent or the dentist there is usually a fistulous opening on the gums in the neighborhood of the root of that particular tooth.

It would not be amiss to say something about pains that are experienced in the mouths of children. Too frequently physicians, sometimes dentists, and nearly always mothers, consider all pain in the teeth of like origin, and therefore, treat it in like manner. Pain in deciduous teeth, or in permanent teeth during childhood is caused by several agencies. There may be a hyperemic pulp as a result of an injury such as a severe bump to a tooth. A child may fall down and strike his tooth against a piece of furniture, the sidewalk or whatnot. That severe injury may not break any enamel off the tooth, and yet the injury may be of sufficient shock to the pulp to cause its death, and if it does cause its death, unless it is treated almost immediately, abscess conditions and infections of the bones about the roots of the teeth occur.

Hyperemia of the pulp is caused by any type of irritant other than infection, that is, we as dentists so classify it. Hyperemia of the pulp is usually a passive hyperemia. There is no swelling, because there is no room for the pulp to swell. It is encased so tightly within the walls of the tooth that if there is an increase in the flow of the blood, there is an increased pressure on the fine nerve endings of the pulp tissue which results in pain. The most severe type of toothache may result from an irritated pulp of traumatic origin, or it may be caused by the use of black copper cement. It may also be the result of the application of such drugs as silver nitrate; the pulp is alive, but is hyperemic and very painful. Then there is inflammation of the pulp which is caused by infection. The pain then is the result of infection within the pulp tissue itself.

There are sometimes abscesses within the pulp tissue; the pulp may be alive and still have an abscess. In that case the pain is from the infected pulp. If the pulp dies the pain is around the tooth and not within it. The parent then goes to the drug store and gets toothache drops, puts them in the tooth or puts it in the cavity of the dead tooth that has an abscess in the pulp tissue itself, which treatment could not be successful.

Dr. Black mentioned the fact that the six year molars usually erupt without pain. Occasionally they do not, however, and with a few such experiences I have had some very peculiar results. The treatment was accidental. I have had teeth roentgenographed for the purpose of determining the cause of the pain, and usually having the roentgen-ray picture taken was sufficient treatment to correct the pain. That happened not only in one case, but in a great many cases where the six year molars were painfully erupting. When the six year molar does come through, very frequently over the distal portion of the tooth, there is a flap of gum tissue, which sometimes becomes infected and very sore. These can best be treated by applying a 15 per cent. solution of silver nitrate under the flap of gum. There are all sorts of referred pains experienced in the mouths of children, particularly those that come from the ears.

Root canal technic in the deciduous teeth has caused considerable discussion in the dental profession. Some men, a limited few, believe in the extraction of every tooth if the pulp is involved. I believe that there is a time when teeth

with dead pulp in them can be treated, and treated successfully, but cases have to be selected very carefully. They should be selected as follows.

There is a period between the time that the deciduous tooth is completely formed and the time that the roots begin to absorb (by natural causes) when root canal work may be tried, if at that time there has been no absorption of the tooth, or considerable absorption of bone as a result of infection. It has been stated that odontoclasts do not cause absorption of deciduous roots, if there is infection about them, and as a result, if the tooth is infected, the roots are not absorbed normally. That is generally true, but there are times when the entire root is absorbed long before the physiologic processes come into play to cause absorption of the root, so before selecting a case for treatment of the root canal a roentgen-ray picture should be taken to show if there has been physiologic absorption or pathologic absorption. If there is a completely formed root, no absorption of bone, it is safe to fill the root canals if the patient is in good health.

There is one tooth in particular that I try to save whenever I can, and that is the second deciduous molar. This is the tooth that keeps the six year molar where it belongs. If the second deciduous molar is lost early the six year molar usually moves forward and results later in faulty occlusion.

Cavities should be filled at any age, even though within six months of the time in which the tooth should be lost, for hygienic reasons. There are several things to be considered. First of all, if there are cavities in the teeth, over any period of time, they are places where food can decay; they are places where bacteria can grow, because they have a very suitable condition for bacterial growth. They have darkness, even temperature, moisture and food. Abscessed, deciduous teeth and roots that cannot be successfully treated should be extracted at once, if the patient's health will permit.

Hollow roots are the finest breeding places for tuberculosis. They cannot be successfully treated and are nurseries for the growth of bacteria. There is no way of fixing them in the mouth so that you can keep them clean. They are a menace to the health of the child and I think they should be out.

DENTAL DEFORMITIES: ETIOLOGY AND TREATMENT. Presented by DR. FREDERICK B. NOYES.

No single item in the human anatomy is of greater importance to the person than is the denture. While both the dentist and pediatrician are concerned with the diseases of childhood, and are brought into close relationship on that account, the common bond between them is the relationship of development. From the standpoint of the orthodontist, one of the difficulties is that the child who needs the pediatrician from the standpoint of the development of his denture may not be desperately ill, and consequently neither the parent nor the pediatrician takes a pronounced interest in him. He needs most careful study but is not likely to get it. One of the greatest difficulties is that the dentist has been interested in the tooth and not the denture, and the unit is the whole denture. The development of the denture is dependent on two things: A mechanical relation of cusps, and their function. If one of the cusps is in wrong relationship the development of the denture is disturbed through its entire extent. However, the cusps may be in their normal relationship but unless the forces of function are in balance, the development of that denture will be proportionately affected.

Many of the problems of the orthodontist are essentially problems of metabolism involving all the factors of growth, and are of as much interest to the pediatrician as to the orthodontist. The first functional disturbance in which they are interested is the function of nutrition in infancy. The normal breast fed baby has to work for his dinner and the amount of muscular exertion that is required develops the muscles. The average bottle fed baby gets his dinner so easily and quickly that there is a disturbance of muscular balance, which is

often the beginning of the disturbance of a normal dentition. Abnormal habits, such as thumb sucking, produce an abnormal pressure, which brings about the result of the abnormal development in the bone and the consequent disturbance of the teeth, and if that continues, it usually produces disturbance in function. One cannot say that every thumb sucking baby will develop malocclusion, but unless the habit is broken in the beginning it is exceedingly difficult to correct. This is the first place in which the orthodontist and pediatrician come into vital contact.

(A series of lantern slides demonstrating the development of human dentition was shown.)